

CLINICAL ASPECTS OF THE ELECTROCARDIOGRAM

Clinical Aspects of the

Electrocardiogram

including the Cardiac Arrhythmias

by

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With 219 Illustrations on 102 Figures

FOURTH EDITION REVISED



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CLINICAL ASPECTS OF THE ELECTROCARDIOGRAM

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D-Q

To

PROFESSOR HORATIO B WILLIAMS

in appreciation of his kindly supervision of the author s introduction to electrocardiography

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PREFACE TO THE FOURTH EDITION

SINCE the publication of the last edition of this book the use of precordial leads has added greatly to the diagnostic value of the electrocardiogram. Their potential value was so evident from the time of the first reports of their use that it was thought advisable to delay the present edition until the groundwork of the new technique had become established. From the beginning it was evident that differences were to be expected between leads from different precordial points and also that when using the same precordial point there would be differences dependent upon the position of the remote electrode. At present we understand the significance of these differences so that instead of finding them confusing we now find them helpful in our diagnosis. The author has attempted to set forth the differences in the record which result from changing the position of the precordial electrode and also the differences resulting from changes in the position of the remote electrode. This discussion indicates the particular value of certain combinations which are recommended for routine use.

For purposes of clinical interpretation it was necessary to establish the character and extent of the variations found in records by precordial leads from different normal hearts. Only with such a background could we say that certain features were normal or were abnormal. Within the last two years this has become possible and condensed tables of the normal measurements of the waves by the most important precordial leads are here included. Several new studies of the form of the electrocardiogram by the limb leads in normal individuals have also appeared during this time so that it has been possible to check our former impressions of the limits of the variations found in records from normal hearts by these leads. For the most part the new work serves to confirm our former ideas.

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The clinical features underlying and causing the abnormalities of the electrocardiogram are amply discussed not only in conjunction with the description of the abnormality itself but also in special sections of the text devoted especially to the clinical aspects This method of approach was considered superior to that of giving case histories in connection with individual records for the latter produces a bulky and cumbersome text containing much matter quite unrelated to the interpretation of the record

A description of the amplifier tube instrument has been added to the chapter on the construction and operation of the string galvanometer This should be helpful to those who are using this increasingly popular type of apparatus and who desire an understanding of its fundamental construction

The bibliography has been greatly expanded and brought down to the present and many of the older references have been omitted because of their diminished value In order to increase the usefulness of the bibliography each chapter is followed by the references pertaining to its subject matter

Acknowledgments are due to Dr Fred M Smith the editor and to the publishers of the *American Heart Journal* for permission to use the section on the nomenclature and description of the electrocardiogram in much the same form as it appeared in this journal Thanks are especially due to Dr Smith for his editorial suggestions in this section

HAROLD E B PARDEE

New York N Y
March 1941

It is not only in the interpretation of precordial leads that progress has been made The knowledge of the abnormalities of the waves produced by various forms of myocardial disease has also greatly increased The new information pertains not only to the effects of rheumatic disease and of coronary arteriosclerosis but also to other less common diseases and metabolic disturbances many of which were not previously known to be associated with electrocardiographic changes When describing the electrocardiographic features associated with each form of cardiac disease the information derived from the precordial leads has been described as an integral part of the record It seemed better to treat the subject in this way than to group all of the information from the precordial leads together as if they were something apart from the limb leads

The chapter on the electrocardiographic diagnosis of cardiac arrhythmias has been expanded and a description of parasystole and of electrical alternans has been included The explanations of the theory of the electrocardiogram are more extensive and have a more practical bearing upon the everyday interpretation of records particularly of abnormal ones The chapter dealing with this subject has been almost entirely rewritten so as to include the newer views but still retains an ample discussion of Einthoven's triangle and its important applications to the interpretation of records The graph devised by Carter Richter and Greene has been reproduced because of its convenience for the determination of the angle alpha from the values recorded in the limb leads

The illustrations in this edition are especially numerous and demonstrate the various features that are described in the text Some of the old illustrations have been replaced by new ones more typical of the conditions and many new illustrations have been added to demonstrate newly recognized abnormalities of the curves or to show additional varieties of those previously recognized Records illustrating the effects of myocardial infarction are especially numerous and also the various types of bundle branch block Each illustration is accompanied by a description of the important features of the curve so that one could acquire considerable familiarity with electrocardiographic interpretation

PREFACE TO THE FIRST EDITION

THE use of electrocardiographic records in the diagnosis of heart disease has been but slowly developed. It was necessary first to determine what the waves of a normal record represented and then to observe a large number of abnormal records in connection with the clinical features of the patients who gave them. Our present knowledge of the interpretation and prognostic importance of the electrocardiogram rests upon such a large number of observations by so many different authors that the beginner is unable to acquire it without consulting a widespread and voluminous literature. I have therefore thought it worth while to collect together in this volume all the current knowledge of the electrocardiogram which is of clinical importance, leaving the details of experimental and theoretical considerations to the special workers in this field. Yet experimental observations and theories cannot be entirely ignored by the clinician, for they give a perspective which clarifies many of his clinical observations. They are therefore included here in as simple a form as possible.

The limits of the normal variations of the electrocardiogram have been carefully described, for only by being familiar with the great variety of these can one distinguish certainly the abnormal curves. The clinical aspect of these abnormal curves has been kept constantly to the fore, that the physician may know what sort of information he may expect to obtain from the records. The electrocardiogram affords but one of the many methods of examining the heart, and though it gives information which cannot be obtained in any other way regarding the contraction of the auricles and ventricles, yet in order to get the full value from the records they must be correlated with the complete history and physical examination of the patient. I have tried to point out the detail of this correlation. Abnormal electrocardiograms

Hoeber and his editorial staff for their interest in the manuscript of this book and for their many helpful suggestions in the matter of arrangement

HAROLD E. B. PARDEE

New York, N. Y.
June 1924

are considered as an expression of abnormal muscle function, and the possibility of an underlying disease is then discussed

The basic electrical and mathematical features of the method have been explained by diagrammatic drawings which may help the reader to obtain the necessary conception of the electrical activity of the heart This technical discussion has been kept almost entirely within the bounds of Chapter IX Perhaps the reader who finds in it an unfamiliar vocabulary will be better prepared to read this chapter after having familiarized himself with the clinical aspects of the records presented in the earlier chapters He will probably find it to be more easily comprehended than he had thought

Controversies have not been discussed in detail The opinion which seems most sound has been presented with a mere statement of the opposing views The references for supplementary reading are intended for those who wish for more detail They do not form a complete bibliography of the subject—this would be too cumbersome to be useful—but give the important standard sources and the best of the newer work

So little has appeared in the literature about the technique of using the electrocardiograph that it was thought worth while to add a chapter on the construction and operation of the instrument Those features which might lead to the taking of incorrect records are especially emphasized and it is pointed out how they may be avoided

It is a pleasure to acknowledge my indebtedness to Professor Horatio B Williams of the Department of Physiology of the College of Physicians and Surgeons Columbia University for his invaluable instruction during my years as his assistant Without the grounding in the fundamentals of electrocardiography received from him the clinical observations of later years would not have been possible I wish also to acknowledge the kindness of Dr Lewis A Conner and Dr W R Williams for the privilege afforded in the study of clinical material in the New York Hospital where most of the records used to illustrate this book were obtained I feel that a final word of thanks is due to Mr Paul B

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CHAPTER I

INTRODUCTION

THE clinical study of the electrical current accompanying the heart s contraction was made possible in 1904 by Einthoven of Leyden through the perfection of an instrument sufficiently sensitive and quick to follow the small rapidly varying potentials produced by the heart It had been known since 1855 that the contraction of the heart was accompanied by the production of electricity In 1887 Waller demonstrated that a current could be led off from the surface of the body and recorded if only a proper contact were made between the wires from the galvanometer and any two areas of the body including the heart between them At that time however the medical world was not prepared to consider seriously so complicated a diagnostic appliance Even the physiologist had difficulty in making use of this knowledge for the only instrument available was the capillary electrometer which owing to its inertia was unable to record with accuracy the quickly varying potentials of the heart beat Einthoven s string galvanometer was first described in 1903 and in 1906 and 1908 he published the results of his first clinical studies The clinical use of the electrocardiograph soon became general in the large clinics abroad and the first instruments were installed in this country between 1910 and 1913

The record of the string galvanometer consists of a series of deflections or waves (Fig 1) produced photographically by the up and down movement of the shadow of the string of the instrument When the patient is connected with the instrument the shadow of the string can be seen to move rapidly in response to the current from the heart which is passing through the patient s body the wires and the string itself These movements are photographed by drawing a strip of film or bromide paper behind a slit through which the light from the galvanometer passes and with the string shadow falling across it (Figs 1 and 93) The pro

Some leads were favorably situated to record the heart s current some leads were unfavorably situated It therefore became neces sary to adopt standard leads in order to facilitate the comparison of records from different patients Leads from the extremities have been universally used for clinical work and have been found satisfactory because of the ease of applying electrodes to the extremities and because the heart occupied a relatively similar position between the extremities in different individuals Only three of the six possible combinations of the extremities were used as follows

Right arm—left arm called Lead 1
Right arm—left leg called Lead 2
Left arm—left leg called Lead 3

Lead 1 is horizontal in relation to the body Lead 2 is oblique and Lead 3 is in the left lateral position and is more or less ver tical This will be clear if we consider the relation of the two shoulders and the left hip to each other One must realize that it is really from the shoulders and hip that the current is led off from the trunk itself (Fig 2)

In each lead the extremity nearer to the apex of the heart is called apical while the other nearer to the basal region is called basal The wires from the extremities are connected to the gal vanometer so that if the apical extremity becomes positive (+) in relation to the basal extremity the resulting current passing through the galvanometer from the apical to the basal extremity in each lead (dotted arrows of Fig 2) will produce an upward movement of the line of the record This standard method of connecting the patient with the galvanometer terminals insures the waves having the same direction in each lead each time the record of the same individual is taken If the wires are reversed the record will be turned upside down If the wires are correctly attached and the waves are changed in their direction it will indicate a change in the potentials within the heart

Precordial Leads

During recent years attention has been called to the impor tance of leads obtained from the precordium Such leads had

gressive movement of the photographic film behind the slit converts the visible movements of the string shadow into the up and down waves of the record An upward wave indicates a current

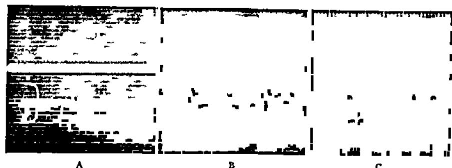


Fig 1 The horizontal and vertical rulings

A The horizontal ruling due to the shadow of the millimeter lines behind the lens The string is at rest and its shadow is straight because it is not now connected to the patient

B The string is now connected to the *patient and shows the movement due to* the electrocardiogram

C The vertical lines are now present due to the shadows of the spokes of the time marker Each division equals 0 04 second and between each accentuated line (due to the wide spoke) the time interval is 0 20 second

in one direction a downward wave a current in another A straight line indicates that no current is passing for the string shadow then remains still as the photographic film moves behind the slit Vertical lines across the record will indicate time because as the paper moves onward successive vertical sections of it lie behind the slit (Figs 1 and 93)

The Three Leads from the Extremities
The Limb Leads

The fundamental fact is as Waller demonstrated that if two wires are connected with two areas of the skin through some appropriate medium such as cloth pads wet with salt solution and these wires are connected to the galvanometer the instrument will record a current if the two skin areas are *both* very near the heart If some distance from it these skin areas should include the heart between them or the current will be very small Any pair of areas from which the current is led off to the instrument is called a *lead* or *derivation* Almost every different lead derived a different variable electrical current from the same heart

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Precordial Leads

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been recorded previously but their clinical value has only been demonstrated since 1932 by the work of Wolferth and of Wilson and their associates and of many other later workers

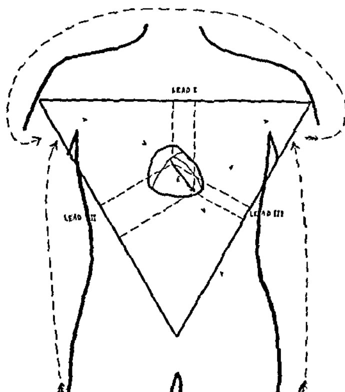


Fig [illegible] Illustrating the relation of the potential within the heart to the current obtained by the three leads The body is viewed from the anterior aspect Each side of the triangle between the right arm the left arm and the region of the legs (left leg) represents one lead

A current resulting from a potential difference within the heart (arrow F) would be represented in each lead by excursions of the same relative size as the projections of this arrow upon the appropriate side of the triangle The line of arrowheads passing through the body from right arm to left arm represents the flow of current in Lead I and the circuit is completed outside the body through the galvanometer as indicated by the long dotted arrow between the two arms The flow within the body is indicated for Lead 2 by the line of arrowheads passing from right arm to left leg The return flow through the galvanometer for Lead 2 is shown by the dotted arrow between the leg and right arm The flow through the galvanometer for Lead 3 is indicated by the dotted arrow between the leg and left arm

When one electrode is placed upon the precordium and the other at some remote point such as the back or one of the extremities the precordial electrode is so much closer to the heart where the potential variations are much greater in magnitude

that the record will be influenced almost entirely by the potential variations of this electrode. The potential variations of a point upon the precordium are much smaller than the potential variations of a point upon the heart but they are still five to ten times as great as those that occur at points on the extremities. These latter potentials rarely exceed 0.5 millivolt for the QRS group and 0.2 millivolt for the T wave. A precordial lead is only a step removed from a direct lead from the surface of the heart itself and has been called by Wilson a "semidirect" lead. In such leads the effect of the activity of the muscle on the anterior surface of the heart which is closest to the electrode will predominate over the effects of more remote portions of the heart, such as the septum or the posterior wall. Because the muscle immediately adjacent to the electrode has the most influence upon it, we shall find that a considerable change in the curve will result from placing the electrode over different points of the precordium.

Leads commonly used involve the application of an electrode to the anterior chest wall over various portions of the heart. One wire is attached to this electrode and the other to an electrode applied either to one of the extremities, to the posterior chest wall mesial to the angle of the left scapula, or to a special connection which brings the three standard extremity wires together at a central terminal of practically zero potential (Chapter IX). Because the potentials at the site of the remote electrode are relatively small, changes in its position have but little influence upon the form of the curve recorded. It has therefore been called by some the "indifferent" electrode. Its position is not, however, entirely a matter of indifference (page 329).

These leads, in which an area of the precordium and some remote area are connected to the galvanometer, have been called chest leads, or better, precordial leads. Because of the confusion which arose from the fact that many different precordial leads were being used by different observers, a joint committee of the American Heart Association and the Cardiac Society of Great Britain and Ireland attempted to standardize the usage of these leads by recommending the routine use of one particular precordial lead. The committee did not intend to discourage the

use of other precordial leads but merely to suggest that this one should always be used *It was suggested that this precordial lead* be called Lead 4 F (F indicating foot) and that the precordial electrode be placed upon the chest wall at the extreme outer border of the cardiac apex as determined by palpation or percussion and the remote or indifferent electrode upon the left leg The wires are to be attached to these electrodes so that the wire which was formerly attached to the apical extremity of either lead would be attached to the precordial electrode and the wire from the corresponding basal extremity would be removed to *the left leg Thus if using the wires of Lead 2 that from the left* leg would be attached to the precordial electrode and that from the right arm would be attached to the electrode upon the left leg * Again uniformity of the attachment of the wires insures that the waves shall always have the same direction when the heart s electricity is the same If the precordial electrode is more positive (+) than the indifferent electrode an upward deflection will result and vice versa

The committee further suggested that the name Lead 4 R be used if the electrode from this precordial point is paired with an electrode on the right arm, Lead 4 L if it is paired with an electrode on the left arm Lead 4 B (B indicating back) if it is paired with an electrode in the left interscapular region and *Lead 4 T if it is paired with a central terminal such as described* in Chapter IX

A further measure to insure the uniformity of precordial electrocardiograms was the recommendation that the precordial electrode should not exceed 3 cm in its greatest dimension A circular electrode between 2 and 3 cm in diameter was specifically recommended The reason for this recommendation is that it has been claimed that a larger electrode would overlie a larger area of the precordium and therefore might in certain special circumstances give rise to a curve different from that which would have been obtained with a smaller electrode

In view of the fact that many might think it desirable to take

* If the wires of Lead 1 are used the left arm wire is to be attached to the precordial electrode and the right arm wire to the leg electrode If the wires of Lead 3 are used the leg wire is to be attached to the precordial electrode and the right arm wire to the leg electrode

leads from more than one part of the precordium the American committee suggested a terminology for records obtained with certain other positions of the precordial electrode as follows *Position 1* to be in the fourth intercostal space at the right margin of the sternum and if the leg is used for the indifferent electrode this lead is to be called CF 1 (chest foot position 1) Position 2 to be in the fourth intercostal space at the left margin of the sternum and the lead from this point paired with the left leg to be called CF 2 Three other positions are designated which lie upon a line drawn from position 2 to the outer border of the apex beat or to the junction of the midclavicular line and the fifth intercostal space if the apex beat cannot be determined and continued around the left side of the chest at this level *Position 3* lies at the left parasternal line *Position 4* at the midclavicular line *Position 5* at the left anterior axillary line and *Position 6* at the left midaxillary line When paired with the left leg these leads are called CF 3 CF 4 CF 5 and CF 6 If these positions of the precordial electrode are used in *conjunction* with the central terminal as described in Chapter IX the letter V (V indicating voltage) substituted for F in the names of the above leads will indicate this fact If they are paired with the right arm the letter R should be substituted for F to indicate the lead

It may be noted that position 4 may sometimes lie just lateral to the cardiac apex and in this case Lead CF 4 and Lead 4 F will be identical (or CR 4 and Lead 4 R) It should be emphasized however that with Lead CF 4 (or CR 4) the point has reference to the bony landmarks of the chest* and with Lead 4 F to the position of the cardiac apex The position of these points will be seen in Figure 3

An orthodiagraphic study of the situation of Positions 1 and 2 (the fourth intercostal space at the right and the left sternal lines) shows that they often lie too high to cover a portion of the main

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body of the ventricular muscle To illustrate this Figure 4 shows orthodiagraphic tracings of six hearts the *numeral 4 indicating in* each case Positions 1 and 2 in the fourth intercostal space Posi

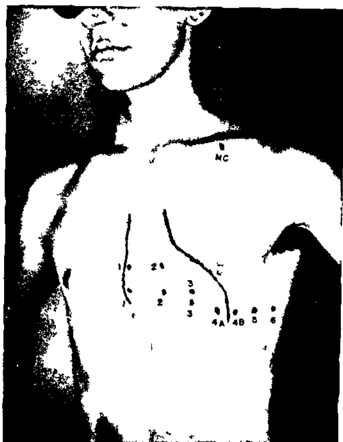


Fig 3 The site of the precordial electrode Positions 1 to 3 as indicated by the Committee of the American Heart Association are seen at the upper points *1 2* and *3* as recommended by the author at the lower points *1 2* and *3* *Positions 4 5 and 6 are the same according to both systems of placing the* electrode The outline of the heart is indicated as if projected orthodiagraphically upon the anterior chest wall The point *4 A* is position 4 as determined by the junction of the midclavicular line and the fifth intercostal space The point *4 B* is the position for the precordial Lead 4 as determined by the location of the apex of the heart It will be realized that if the heart were not enlarged *4 A* and *4 B* might coincide The midclavicular point is indicated at *MC*

tion 1 is seen to lie over the extreme basal portion of the right ventricle or frequently not over the heart at all but over the aortic arch or completely to the right of the mediastinum over the margin of the lung The area of the right ventricle beneath Position 2 is the outflow portion of this chamber or the pulmon

ary conus and is an area which is not commonly affected by disease particularly not by coronary arteriosclerosis The numeral 6 however which is in the left sternal line at the level of the

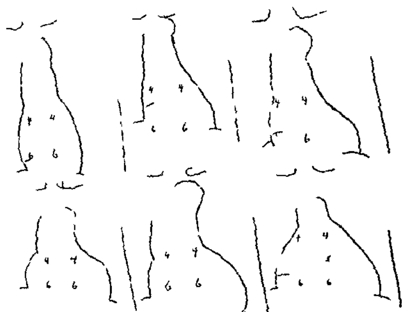


Fig 4 Orthodiagraphic tracings to show the position of the junction of the fourth and sixth intercostal spaces with the sternum in relation to different types of cardiac configuration The sixth intercostal-sternal junction is opposite the upper end of the ensiform

upper end of the ensiform lies over a portion of the ventricular myocardium much more frequently affected by disease A corresponding point below Position I in the right sternal line will often overlie the right auricle but in other cases will overlie a portion of the right ventricular myocardium as indicated by the numeral 6 on the right of the midline in Figure 4 Reference also should be made to Dressler's determination of the portions of the heart underlying various parts of the precordial area His illustrations also show that the fourth intercostal space is usually too high to overlie important areas of the ventricular myocardium

Figure 5 A is a record from the heart giving Figure 4 F the upper lead being from the fourth left interspace at the sternal

line (CF 2) and the lower being in the left sternal line at the level of the upper end of the ensiform In this record T is diphasic (+—) in Lead CF 2 but not in the lead from opposite

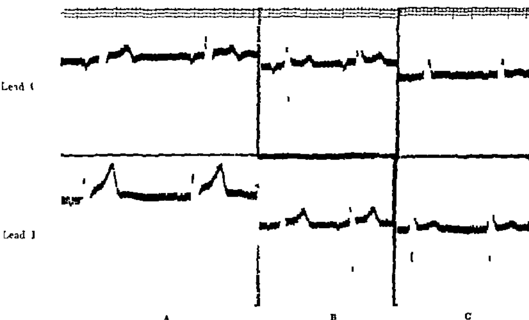


Fig 5 Upper line records from fourth interspace to left of the sternum Lower line records from the left sternal border at the level of the sixth interspace or ensiform cartilage

A shows differences in the height of the waves of the QRS group and of T T is diphasic (+ —) in Lead CF 2 and large and upward in the lead from the left of the ensiform

B shows marked differences in the form of the QRS group in the two positions and some difference in the amplitude of T

C shows differences in the form of QRS in the two positions CF 2 shows a very small T wave while from the level of the ensiform the T wave is definitely upright

the ensiform The other records show differences between the leads from these two points but these differences are not so suggestive of an abnormal curve as is the diphasic T wave

Since the muscle areas of the ventricles lie nearer to the diaphragm it is suggested that points opposite the ensiform in the right (E 1) and left (E 2) sternal lines should be more useful than points as high as the fourth intercostal space and this lower level has been used instead of the fourth interspace in obtaining the records used as illustrations in this volume The designations EF 1, EF 2 and EF 3 may be applied to the leads from positions at the level of the ensiform the last being below the position of

CF 3 of the Heart Committee The different positions of these points will be apparent in Figure 3

It is important to emphasize that these orthodiagraphic tracings were made with the patient in the erect position With the patient recumbent the heart moves upward in the chest so that the various points on the chest wall thereby become related to different parts of the anterior surface of the heart This vertical displacement of the heart with recumbency is accompanied by a displacement posteriorly and probably by a slight rotation of the heart on its long axis so that these features contribute to the presentation of different parts of the heart beneath any fixed point of the precordium when the patient changes from an upright to a recumbent position or vice versa This matter has been studied in a few cases by Stewart and Bailey who found that there was usually some change in the waves of the precordial electrocardiogram when the patient changed from the recumbent to the sitting position the most frequent change being a decrease in the amplitude of the T wave The amplitude of R also usually decreased but S although usually varying with position failed to show a definite tendency

Experience has shown that in many patients more than one precordial point must be used in order to obtain evidences of myocardial disease Leads from the following points are recommended by the author to be taken routinely

1 The left sternal line at the level of the ensiform (this lies below Position 2 of the Heart Association and the lead may be called Lead FF 2)

2 A point halfway between the first point and the next (this lies below Position 3 of the Heart Association and the lead may be called Lead EI 3)

3 Just lateral to the cardiac apex (this corresponds to the position of Lead 4 F of the Heart Association Lead 4 F)

4 The left anterior axillary line at the level of the cardiac apex (this corresponds to Position 5 of the Heart Association Lead CF 5)

5 If the heart is enlarged to the anterior axillary line an additional lead should be used from the midaxillary line at the level

line (CF 2) and the lower being in the left sternal line at the level of the upper end of the ensiform In this record T is diphasic (+—) in Lead CF 2 but not in the lead from opposite

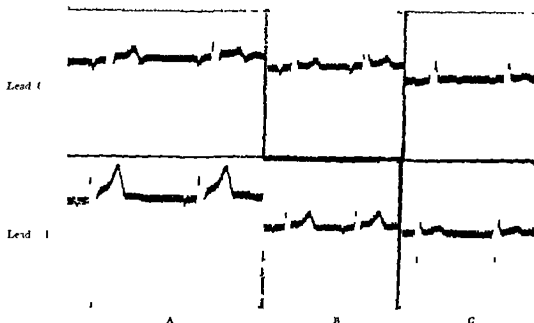


Fig 5 Upper line records from fourth interspace to left of the sternum Lower line records from the left sternal border at the level of the sixth interspace or ensiform cartilage

A shows differences in the height of the waves of the QRS group and of T T is diphasic (+ —) in Lead CF 2 and large and upward in the lead from the left of the ensiform

B shows marked differences in the form of the QRS group in the two positions and some difference in the amplitude of T

C shows differences in the form of QRS in the two positions CF 2 shows a very small T wave while from the level of the ensiform the T wave is definitely upright

the ensiform The other records show differences between the leads from these two points but these differences are not so suggestive of an abnormal curve as is the diphasic T wave

Since the muscle areas of the ventricles lie nearer to the diaphragm it is suggested that points opposite the ensiform in the right (E 1) and left (E 2) sternal lines should be more useful than points as high as the fourth intercostal space and this lower level has been used instead of the fourth interspace in obtaining the records used as illustrations in this volume The designations EF 1 EF 2 and EF 3 may be applied to the leads from positions at the level of the ensiform the last being below the position of

taken in the erect and recumbent positions the record in each case being obtained with the electrode over the same point on the bony thorax

Esophageal Leads

The use of leads from the esophagus has been revived recently and several clinical studies of normal individuals and of patients have been published Thus far the method has been too little used to demonstrate its value for clinical investigation It is evident however that it will produce semidirect curves from the left auricular muscle and from the basal part of the left ventricle neither of which areas are otherwise available to semidirect leads It should be capable of demonstrating abnormality in these muscle areas better than leads from the anterior chest wall

It has been shown that when the esophageal electrode lies in the region of the auricles the auricular deflection contains rapid vibrations analogous to the QRS group of the precordial leads These deflections are not present when the esophageal electrode is not over the auricular muscle Leads from below the levels at which such auricular waves are obtained are probably leads from the posterior basal portion of the ventricles at first but as the electrode goes lower it eventually leaves close contact with the heart as does the precordial lead from the anterior axillary line (Lead CF 5)

The Nomenclature of the Waves

Figure 6 is an electrocardiographic record taken with the string galvanometer by the usual three limb leads The leads are not taken simultaneously as might appear from the illustration but one after another the whole process consuming from three to five minutes The series of steps at the left end of each lead is the control of the standardization Each step is due to a difference of 1 millivolt of potential applied by the operator in the circuit with the patient and will be seen to measure 10 mm * The position of the string shadow during diastole is called the base line or zero level and there are groups of waves (deflections) pointing

* Upward deflections are measured from top of the base line to the top of the wave downward deflections from the bottom of the base line to the bottom of the wave

of the cardiac apex (this corresponds to Position 6 of the Heart Association, Lead CF 6)

These will cover areas of the myocardium sufficiently remote *from each other so that occasionally the record from only one or* two of them will show a significant abnormality For this reason the author recommends that the first four of these points should be routinely used and the fifth point also in the presence of marked cardiac enlargement

It is recommended also that the indifferent electrode shall routinely be placed upon the left leg because more normal control records have been made by this technique and because it has been shown by Geiger to give an abnormal curve more frequently than leads using the right arm In the majority of cases if their position is checked orthodiagraphically the position of the electrode for Leads FF 2 and FF 3 will be found to lie over different portions of the right ventricle *Occasionally the position* for Lead 4 F will lie very close to the left border of the heart usually *close to the cardiac apex but may be somewhat lateral* to it or above it or both above and lateral The first two of these points will bear a closer relation to the chambers of the heart with the varying chest configuration and heart size encountered in different patients than the second and third positions recommended *by the American Heart Association which are deter* mined by landmarks on the bony thorax alone Depending upon whether the heart is vertical or transverse small or large these latter positions may find quite different parts of the myocardium beneath them in different patients

In women when there is an excessive amount of breast tissue difficulty may be experienced in applying the precordial electrode especially if the record is obtained with the patient in the sitting position It is probably better to attempt to place the electrode high up under the breast so that it comes in the fifth intercostal space Records taken with the electrode over the same part of the fifth intercostal space with the electrode upon the breast are not materially different as a rule though the excursions of all waves are usually somewhat smaller The author has not observed as much difference between records taken through and beneath the breast in the same individual as between records

Each group of waves represents one complete cardiac contraction and is called an electrocardiogram. The letter placed opposite the peak of each of these waves has been adopted as the conventional name for the wave but it is not to be implied that waves of like name will always be due to the same activity of the cardiac cycle. This is usually the case for the peaks of the P waves in the limb leads and is often the case for the peaks of T but it is not usual for the peaks of the QRS group to represent the same phase, that is, the same instant, of cardiac activity (page 352). Each heart cycle is ushered in by a small rounded elevation called P. This is due to the activity of the auricular muscle. The P wave is followed by a series of waves due to the activity of the ventricular muscle. First there is a group of sharp pointed upward and downward deflections called the QRS group. If there is an upward deflection it is called R; if a downward deflection precedes R it is called Q; if a downward deflection follows R it is called S; when no R is present, a downward deflection is to be called QS.* At times there are more than three deflections in the QRS group or the deflections may not follow the usual pattern. There may be an upward deflection followed by a downward and then another upward deflection making a sort of M shaped complex (Leads CF 2 of Fig. 5 B and FF 2 of Fig. 5 C). Occasionally there are as many as two or three upward and two or three downward deflections producing what might be called a vibratory type of QRS group (Lead 3 of Fig. 10). In certain abnormal records the largest deflection of QRS may be downward in one or more leads. This is called Q or S according to its relation to the upward deflection of the respective lead or is to be called QS if there is no upward deflection.* It is not usual to speak of an inverted or downward R wave in discussing clinical electrocardiograms. (See Chapter IX for a further discussion of nomenclature.)

Following the QRS group the movements become slower. The line of the record appears to rest briefly at or very close to zero and then curves upward or downward from this point toward the

* In order to avoid confusion it is necessary to point out that previously such waves have been called S by the author and Q by Wilson and his associates. The term QS is now suggested as a compromise to avoid the confusion of a conflicting terminology and for other reasons detailed on page 3[illegible].

upward and downward from this base line The zero level is usu-ally horizontal as in Lead I of Figure 6 though it often varies (wanders) somewhat either upward or downward during the tak-

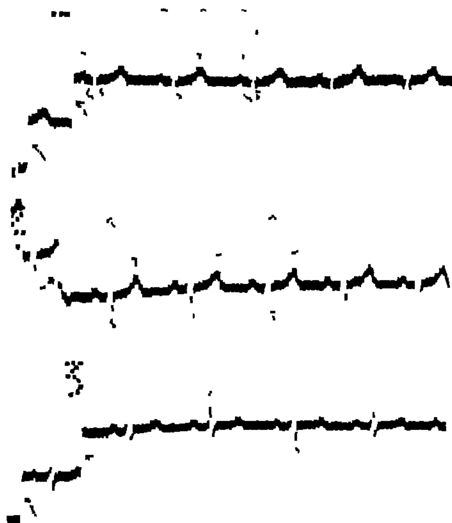


Fig 6 Electrocardiogram of a normal individual taken by the three limb leads In all records the horizontal lines represent strength of current one division = 0 1 millivolt (0 0001 volt) The vertical lines represent time units and when forming a system of small squares as in this record each line represents 0 04 second

The series of steps indicated by arrows at the left end of each lead is a test *of the standardization of the instrument performed by the operator as described* in the text (page 382)

ing of the record as it does during the last two heart cycles of Lead I and throughout the whole of Lead 2

Each group of waves represents one complete cardiac contraction and is called an electrocardiogram The letter placed opposite the peak of each of these waves has been adopted as the conventional name for the wave but it is not to be implied that waves of like name will always be due to the same activity of the cardiac cycle This is usually the case for the peaks of the P waves in the limb leads and is often the case for the peaks of T but it is not usual for the peaks of the QRS group to represent the same phase that is the same instant of cardiac activity (page 392) Each heart cycle is ushered in by a small rounded elevation called P This is due to the activity of the auricular muscle The P wave is followed by a series of waves due to the activity of the ventricular muscle First there is a group of sharp pointed upward and downward deflections called the QRS group If there is an upward deflection it is called R if a downward deflection precedes R it is called Q if a downward deflection follows R it is called S when no R is present a downward deflection is to be called QS * At times there are more than three deflections in the QRS group or the deflections may not follow the usual pattern There may be an upward deflection followed by a downward and then another upward deflection making a sort of M shaped complex (Leads CF 2 of Fig , B and EF 2 of Fig 5 C) Occasionally there are as many as two or three upward and two or three downward deflections producing what might be called a vibratory type of QRS group (Lead 3 of Fig 10) In certain abnormal records the largest deflection of QRS may be downward in one or more leads This is called Q or S according to its relation to the upward deflection of the respective lead or is to be called QS if there is no upward deflection * It is not usual to speak of an inverted or downward R wave in discussing clinical electrocardiograms (See Chapter IX for a further discussion of nomenclature)

Following the QRS group the movements become slower The line of the record appears to rest briefly at or very close to zero and then curves upward or downward from this point toward the

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peak of the final deflection called T The curve which forms this *initial limb of T usually is concave toward the base line* so that it is concave either upward or downward depending upon whether the peak of T is directed upward or downward The peak of T is followed by a return of the record toward the base line This portion of T is of shorter duration than the initial limb of T and because of this the peak of T lies toward the latter part of the wave and the final limb may at times be almost a straight line at first though ending with a slow curving fusion with the horizontal A broad low wave called U is usually observed following T It is possibly due to the anabolic activity of *the heart but its cause is not definitely understood After the U* wave or if U is not present after the end of T the line of the record remains at zero during the diastole of the heart until the next P wave

Though P and T are usually upward deflections as just described this is not always the case In certain normal records and especially in abnormal records either or both of these waves may *be so small as to be almost indiscernible in one lead in which* case the wave is said to be of low amplitude or isoelectric for example the T wave of Lead 3 in Figure 58 D If P or T are found to be turned downward they are often spoken of as being inverted or as a downward P or T wave Occasionally these waves may show both a downward and an upward peak and such a wave is called a diphasic P or T wave (T is diphasic in Lead 3 of Fig 76 A to F) *If a diphasic wave has its initial deflection downward* and its final deflection upward it may be described as diphasic —+ if its initial deflection is upward and its final deflection is downward it may be described as diphasic +— These variations may be normal or abnormal as will be described later If it is desired to speak of a wave in a certain lead this may be accomplished by placing the number of the lead after *the name of the wave so that P_1 means the P wave of Lead 1 R_2* the R wave of Lead 2, and so on

The Amplitude of the Deflections and the Voltage of the Wave

In order that the amplitude of the deflections of different

vanometer so that the deflection resulting from a standard current shall be of a standard size The technique of the standardization is described in detail in Chapter X It will suffice the clinician to know that in every properly taken record a deflection of the string shadow for a distance of 1 cm means that there has been a change of 1 millivolt of potential in the galvanometer circuit Currents of different strengths are represented by deflections proportionately larger or smaller Records should include in each lead a control curve of the standardization for by an inspection of this both the exactness of the instrument and the technique of the operator can be determined (Chapter X) At times the deflections of the precordial leads will be so large that it will be necessary to reduce the standardization In such records the standardization curve should be recorded routinely and it is suggested that one half of the normal standardization always should be used that is 0 5 cm deflection = 1 millivolt

With the leads from the extremities we must consider separately the amplitude of the deflections of each of the three chief parts of the record the P wave the QRS group and the T wave One of the three leads may be unfavorably situated for recording one of these parts of the electrocardiogram and its deflection will be small in that one lead The other two leads however will show larger deflections giving a satisfactory indication of the true value of the potential of this portion of the record for example in Figure 11 C compare the T wave of Lead 3 with that in Leads 1 and 2 Owing to the triangular arrangement of the three leads about the heart only one lead can be unfavorable for any one of the three parts of the electrocardiogram in a given patient (Chapter IX)

In leads from the extremities the amplitude of each of the three parts of the electrocardiogram will usually be found largest in one lead not so large in the other two At times the excursions may be equally large in two leads while the other lead will show a very small excursion or perhaps none at all This is the case with the P wave of Figure 11 A the T wave of Figure 11 C and the QRS group of Figure 11 C and II The size of the largest excursion recorded in any one of the three leads is the best indica

tion of the *voltage* within the heart giving rise to that portion of the electrocardiogram *

The voltage within the heart is always short circuited in its passage to the extremities and in this each of the three parts of the record P QRS and T will be affected equally Certain things near the heart such as voluminous lungs pericardial fluid and possibly pericardial fat have an especially marked effect in re*ducing the voltage and therefore the amplitude of the deflections* in the three leads from the extremities The precordial leads however are not subject to short circuiting in the same way as the leads from the extremities and therefore their deflections may be of normal size (amplitude) at times when the voltage in the limb leads may be quite small as shown by low amplitude in all three leads It will be seen that the term voltage as used here is only to be applied to the leads from the extremities

That waves which are due to an identical potential within the heart may have *different amplitude in the three limb leads* is due to the fact that the direction of the potential within the heart producing the waves has a different relation to the direction of each lead A lead whose direction is approximately parallel to the direction of the potential will give a good representation of the voltage a relatively large amplitude of the wave This is the favorable lead for that potential A lead whose direction is approximately at right angles to the direction of the potential within the heart will give but a small amplitude of the wave no matter how large the potential This may be called an unfavorable lead for that potential One lead may be favorable for one part of the electrocardiogram and not for the other parts Thus in Figure 11 A Lead 3 is unfavorable for P but favorable for QRS and for T while in 11 G Lead 3 is unfavorable for QRS and for T but favorable for P In Figure 16 Lead 3 is favorable for QRS but unfavorable for P and for T

The reader will realize that the amplitude of a wave in its largest lead is a better indication of the potential developed during that part of the heart cycle than the height of this wave in its less favorable leads Low amplitude or flat waves in one lead may be

* The voltage as here defined corresponds roughly to what Einthoven has called the manifest value or Em (page 410)

taken to indicate an intermediate position between an upward and a downward deflection in that lead. This distinction in the limb leads between the low amplitude of a wave in one lead and low voltage of that wave as determined by the three leads is a very important one. *Low voltage* waves are manifested by low amplitude in all leads and indicate a diminished production of electricity within the heart or a greatly increased short-circuiting of the heart's current before it reaches the extremities.

That a wave may have a *different direction* in one or more limb leads is due to the fact that the current producing it may flow toward the apical extremity of one lead and toward the basal extremity of another lead, so that in the first case an upward deflection results and in the second case a downward deflection; for example, the T wave of Figure 11 A is upward in Lead 1 and Lead 2 and downward in Lead 3.

Amplitude of Deflections in Precordial Leads

The size of the deflections in the precordial leads is not related to the size of the same wave in leads from the extremities. Being due for the most part to the potentials produced by the portion of the heart lying immediately beneath the precordial electrode, these deflections are larger than the voltage of the leads from the extremities and vary independently of it.

Deflections Not Due to the Heart

Two rather common causes of distortion of the record must be mentioned in order to point out that they are independent of the action of the heart. One is seen as a fine vibration of the line of the record in Figure 27 B and in Lead 1 of Figures 23 A and 28 A. This at times may be so marked as to obscure the waves of the electrocardiogram. It is due to the tonic activity of the voluntary muscles of the *extremities*, though not to actual movements of the limb. If the arm or leg is held stiffly, as with a patient who is apprehensive, or if the body or the limb is cold and the shivering reflex evoked, these vibrations will be very noticeable. Satisfactory records of patients with tremor or with chorea may be very difficult to obtain.

The other type of distortion already noted in Figure 6 is seen also in Figure 49 B It consists of an upward or downward drifting movement of the base line of the record This slow drifting movement is due to the reflex vasomotor effect upon the skin vessels of a varying mental state The skin change causes a change in the electric potential of the limb in some way which is not understood, and this causes the wandering movement of the base line With a nervous patient, this movement may be more rapid and may be almost continuous causing the base line to move continually over a range of 5 to 10 mm or more, and thus make it very difficult to standardize the instrument before taking the record

SOURCES OF ERROR IN TECHNIQUE

At the beginning of the record of each lead in Figure 6 is the control of standardization The base line of the record is suddenly deflected upward or downward for ten scale divisions which in the original record are each 1 mm This is done by the operator who turns in or out a potential of 1 millivolt (0 001 volt) in the electric circuit containing the patient and the galvanometer The resulting deflection should be exactly 1 cm should be completed in 0 02 second or less and should not show any overshooting of the string before finding its new level (For a discussion of overshooting see Chapter X) If these requirements are not complied with by the control it indicates an error in technique which will result in the record being an inexact picture of the electrocardiogram of the patient It is a less serious error in technique if the deflection of the control should fail to be 1 cm than if the deflection should take longer than 0 02 second or if there should be overshooting (page 387) The first can easily be corrected by adding to or subtracting from the height of the wave in the record the percentage error in the control for example if the control deflection is only 8 mm instead of 10 mm then each deflection in the record should be 25 per cent larger than it is A slow deflection time or the presence of overshooting in the deflection will result in a distortion of the record for which it is very difficult to make proper correction

Physiological Origin of the Waves

The cardiac muscle produces this complicated series of waves with each heart beat because it is of complex structure and stimulated to contract in a complicated fashion To understand these waves and their variations we must understand the anatomical structure of the heart and particularly the mechanism responsible for distributing the stimulus that initiates the contraction of the muscle fibers

The auricles contract in response to a stimulus that is formed in the sino auricular node This is about 3 cm in length and lies in the wall of the right auricle just below and in front of the entrance of the superior vena cava (Fig 7) The stimulus spreads radially from this node throughout the auricular muscle involving the two chambers in contraction almost synchronously Arriving at the auriculoventricular node at the base of the interauricular septum the contraction produces an effect upon the node which starts an impulse along the auriculoventricular bundle (bundle of His) toward the ventricles It is possible that the fibers of the bundle of His may also contract but they do not produce a potential which is demonstrable in the clinical electrocardiogram

Coursing along the top of the interventricular septum the bundle of His divides sending one branch down into either ventricle The one to the right side runs in the wall of the septum toward the septal papillary muscle of this ventricle comes to the surface of the endocardium below this and branching widely breaks up into a fine network over the inner surface of the ventricle A rather large branch passes to the anterior papillary muscle The left branch of the bundle enters the left ventricle beneath the right cusp of the aortic valve and lying superficially beneath the endocardium of the left side of the septum where it is usually visible promptly divides into two main branches One of these passes toward the base of each of the papillary muscles of the left ventricle sending off many branches on the way which spread and break up into a network over the inner surface of the ventricle The ramifications of the subendocardial

branches in each ventricle penetrate the ventricular wall seeming to follow the main muscle bundles and eventually to make contact with the muscle fasciculi These terminal ramifications

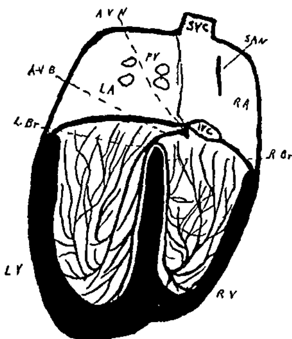


Fig 7 A diagrammatic view of the heart from behind and somewhat below The superior vena cava (*SVC*) and the inferior vena cava (*IVC*) are seen entering the right auricle The mouths of the pulmonary veins (*PV*) are seen entering the left auricle The sino auricular node is indicated (*SAN*) The ventricles are represented as if cut open from behind so as to show the auriculoventricular node (*AVN*) and the auriculoventricular bundle (*AVB*) of His and its branchings in the right and left ventricles *L Br* left branch *R Br* right branch The pentration of the ramifications into the walls of the ventricles is not indicated

have been demonstrated within one or two millimeters of the pericardial surface of the ventricular myocardium

The spreading of the contraction in the auricles is comparatively slow and wavelike and gives rise to the P wave By the time that P has returned to the base line the contraction has spread to involve the entire auricular muscle Auricular relaxation is not completed however until from 0 30 to 0 10 second after P has begun as is indicated by the ending of the slight downward deflection following P seen in Figures 52 A and B This deflection following P has been called the auricular T wave It

is responsible for the difference between the level of the base line of the record preceding P and that of the interval between the ending of P and the beginning of the QRS group The termination of the auricular T wave is only visible when the conduction time is markedly prolonged or when auriculoventricular block is present for coming as it does during the upstroke of the T wave its ending usually is obscured

The delay in the passage of the impulse from auricles to ventricles seems to occur at the auriculoventricular node The tissues of the auriculoventricular bundle and its ramifications in either ventricle conduct the impulse very rapidly to the muscle fibers of the two ventricles The finer branches of these ramifications are known as Purkinje tissue and pass into the ventricular wall following the muscle bundles for an unknown distance before making contact with the fibers As has been said the course of the Purkinje fibers has been traced to within one or two millimeters of the pericardial surface of the ventricles The subpericardial layers of the muscle are the last to be involved in the contraction and those fibers near the base of the left ventricle are the last of these The whole ventricular muscle thus enters into contraction very rapidly considering its size because it is stimulated almost simultaneously at so many widely separated points by means of this complicated mechanism

While the contraction is spreading throughout the ventricular muscle the QRS group is inscribed When the spreading has involved all of the ventricular fibers the QRS group is completed The time taken for the spreading of the contraction throughout the ventricles is about the same as was needed for the wavelike spreading throughout the much smaller auricular mass During this time the electrical production is continually varying in its aggregate potential and its summated direction within the heart and these variations give rise to the quickly changing size and direction of the peaks of the QRS group

With the whole of the ventricular muscle contracting and the quick deflections of the QRS group completed the line of the record rests briefly at or very close to zero This indicates an approximate electrical balance at the junction between QRS and

T, and marks the beginning of the T wave The T wave develops slowly during the ventricular contraction reaches its peak and then returns more quickly to the base line ending just before

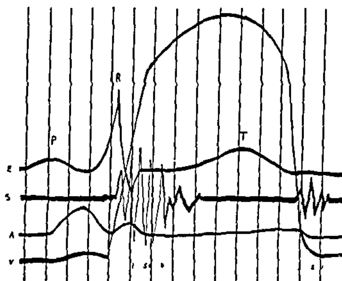


Fig 8 Simultaneous records of the electrocardiogram (*E*) the heart sounds (*S*) and the pressure changes within the right auricle (*A*) and the left ventricle (*V*) These are represented within the same system of time lines as if obtained from the same heart beat each line representing 0 01 second

Note that the P wave of the electrocardiogram begins before the rise of pressure within the auricle and QRS group before the rise of pressure within the ventricle and slightly before the beginning of the first heart sound The end of T slightly precedes the second heart sound and the sharp drop of intraventricular pressure

the time of occurrence of the second heart sound (Fig 8) at which time the ventricular contraction has ended

A detailed explanation of the causes of electrical production during the cardiac cycle is not yet possible though many hypotheses have been advocated and abandoned In the light of present knowledge however we seem in a position to explain many of the underlying electrophysiological phenomena and these are discussed in Chapter IX

Figure 8 has been constructed to indicate the time relations of the waves of the electrocardiogram to other physiological activities of the heart It shows within the same system of time lines the electrocardiogram a record of the heart sounds as if taken simultaneously by means of a microphone and a second galvanometer, a curve of the pressure variations within the right au

tricle and a curve of the pressure variations within the left ventricle The curves of this diagram have been properly placed within the time lines by reference to such synchronously taken records as could be found in current physiological literature It must be remembered that the auricles and ventricles do not contract and relax as a single unit first the two auricles contract more or less simultaneously and then after a slight pause the two ventricles Even within these major units certain fibers receive the stimulus (the excitation) before others and these begin their contraction before the others This relatively gradual onset of the contraction of the muscle of different parts of the wall of the cardiac chambers causes the rise of pressure within to lag slightly behind the change in electrical potential which is the first evidence of muscle activity It is for this reason that the P wave begins before the rise of pressure within the auricles and the QRS group before the rise of intraventricular pressure Furthermore the beginning of the fall of pressure within the cavity does not indicate the ending of the muscular activity but only a predominance of the relaxing muscle fibers over those that have not yet relaxed The electrical curve therefore continues as the descending limb of T after the pressure begins to fall

The P wave of the electrocardiogram must be referred to the beginning of the activity of the muscle fibers of the walls of both auricles since these chambers contract almost simultaneously The P wave begins before the rise of pressure within the auricle and is completed before the auricular contraction has reached its end The QRS group produced by the beginning of activity of the muscle fibers in both ventricles begins before the rise of intraventricular pressure and before the first heart sound The marked difference between the form of the P wave and of the QRS group is dependent upon the different manner in which the contraction stimulus is distributed to the muscle of the auricles and of the ventricles

The T wave reaches its full development during the rise of intraventricular pressure its peak approximately coinciding with the highest point of the pressure curve It falls when the pressure curve is falling but more abruptly and reaches the base line at about the time of the sharp drop of intraventricular pressure

that goes with closure of the aortic valves The second heart sound begins with or just after the end of the T wave as it reaches the base line From its position in the heart cycle the T wave is obviously related to the full development of the ventricular contraction and to the beginning and ending of ventricular relaxation

Measurement of Waves

The *height* of a wave should be measured from the zero level of the record either upward or downward according to the direction of the wave When measuring upward we should start from the top of the shadow of the string and measure to the top of the deflection when measuring downward we should start from the bottom of the shadow of the string and measure to the lowest point of the deflection In measuring the *duration* of a wave it is well to find one which leaves the base line synchronously with one of the time lines We may then count the number of time lines which occur before the deflection returns again to the zero level The wave should be measured in all three leads and the lead giving the largest measurement of its duration should be accepted for sometimes either the beginning or the end of a wave may be unfavorably recorded in one or two of the leads so that its full duration does not appear Figure 9 illustrates this procedure diagrammatically It will be noted that the Q and S waves are not measured from the lower edge of the base line but from the lower edge of the string shadow at the end of the P R interval Likewise the R wave is measured from the upper edge of the string shadow at the same point This is done because the P R interval shows a steady deflection of the base line due to the auricular T wave upon which are superimposed the potentials producing the QRS group The difference resulting from the use of the base line or the P R level to measure the height of these waves is usually only a slight one so that ordinarily it can be disregarded and the base line of the record can be used This is especially so if the wave to be measured is a large one The smaller the deflection of Q or R or S the greater the importance of using a correct point from which to measure these waves

In measuring the duration of a wave whose beginning or ending is gradual as is common with the P and T waves it is sometimes difficult to decide at which points the measurements

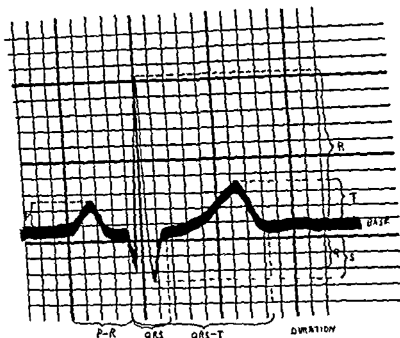


Fig 9 The technique of measurement of the height of the waves and of their duration The brackets below the figure indicate the duration of P R QRS and of the whole ventricular complex QRS T P R equals 0 1C second QRS equals 0 10 second QRS T equals 0 37 second The vertical brackets indicate the measurement of the height of the waves P equals 1 5 mm Q equals 2 mm R equals 9.. mm T equals 2 6 mm

It will be noted that the height of P R and T is measured upward from the top of the base line the height of Q and S is measured downward from the P R level If R were a small deflection it would have been necessary to measure it from the P R level also (see text)

shall be made Careful inspection preferably with magnification should be used to determine exactly where the string shadow makes its first departure from the base line This can only be done exactly when there are no artifacts deforming the base line It cannot in many cases be decided upon within less than 0 01 second but except in the duration of QRS it is not usual for differences of such magnitude to be of importance If they become so it is advisable to measure several heart cycles

that goes with closure of the aortic valves. The second heart sound begins with or just after the end of the T wave as it reaches the base line. From its position in the heart cycle the T wave is obviously related to the full development of the ventricular contraction and to the beginning and ending of ventricular relaxation.

Measurement of Waves

The *height* of a wave should be measured from the zero level of the record, either upward or downward according to the direction of the wave. When measuring upward we should start from the top of the shadow of the string and measure to the top of the deflection; when measuring downward we should start from the bottom of the shadow of the string and measure to the lowest point of the deflection. In measuring the *duration* of a wave it is well to find one which leaves the base line synchronously with one of the time lines. We may then count the number of time lines which occur before the deflection returns again to the zero level. The wave should be measured in all *three leads and the lead giving the largest measurement of its* duration should be accepted, for sometimes either the beginning or the end of a wave may be unfavorably recorded in one or two of the leads so that its full duration does not appear. Figure *9 illustrates this procedure diagrammatically. It will be noted* that the Q and S waves are not measured from the lower edge of the base line but from the lower edge of the string shadow at the end of the P-R interval. Likewise the R wave is measured from the upper edge *of the string shadow at the same point.* This is done because the P-R interval shows a steady deflection of the base line due to the auricular T wave upon which are superimposed the potentials producing the QRS group. The difference resulting from the use *of the base line or the P-R* level to measure the height of these waves is usually only a slight one so that ordinarily it can be disregarded and the base line of the record can be used. This is especially so if the wave to be measured is a large one. *The smaller the deflection of Q or R or* S the greater the importance of using a correct point from which to measure these waves.

dicate the points at which the beginning and ending of P QRS and T are to be measured and the respective measurements are indicated below Records taken at the standard speed should be afforded a similar treatment and as has been stated preferably with magnification by a hand lens

Examination of the Record

When examining an electrocardiographic record the following features of each wave should be noted in each lead

1 The direction of the deflection
2 The height
3 The duration as measured by its straddle on the base line
4 The form (sharp rounded peaked diphasic notched etc)

In addition there should be noted in the leads from the extremities

5 The height of the wave in the lead giving the largest excursion (voltage)

It will be helpful for the reader to turn at this time to the section on How to Read an Electrocardiographic Record (page 249) Although a few terms may be encountered which are not yet familiar this section will give an idea of the procedure to be used when inspecting the illustrations which will follow

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and select those measurements which are longest from the cycles *having the most definite transition from the base line to the* wave.

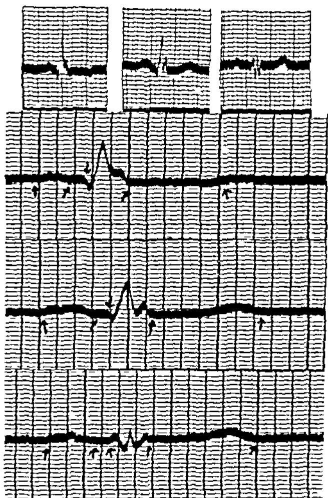


Fig. 10. Above are Leads 1, 2 and 3 at the standard film speed of 25 mm. per second. *Below are the same leads taken with the camera film moving at 100 mm.* per second. Note the notching of R 1 and R 2 and the vibratory QRS 3. The arrows in the records at 100 mm. point to the beginning and ending of the P wave, the beginning and ending of QRS, and the end of T.

In Lead 1 the duration of P = 0.08 second, P-R = 0.12 second, QRS = 0.10 second, Q-T = 0.30 second.

In Lead 2 the duration of P = 0.12 second, P-R = 0.15 second, QRS = 0.10 second, Q-T = 0.34 second.

In Lead 3 the duration of P = 0.11 second, P-R = 0.14 second, QRS = 0.10 second, Q-T = 0.34 second.

Figure 10 shows an electrocardiogram taken at a speed of 100 mm. per second which is four times the normal. The arrows in

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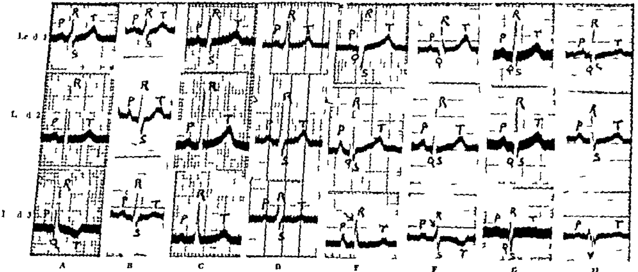


Fig. 11. Eight normal electrocardiograms to show the variations which may be encountered. In each case the upper electrocardiogram is by Lead 1, the middle one by Lead 2, and the lower by Lead 3.

Note the variations in the relative height of R in Leads 1, 2 and 3 as we pass from records A to H. Lead 2 always has the largest value. Lead 1 and Lead 3 vary *reciprocally*.

The fine vibrations of the line of the record, best seen in Lead 3 of B and C, are due to the electrical activity of the striate muscle of the extremities. The patient was not sufficiently relaxed. The blurring of the line of the record in B, F and H is due to the string shadow not being sharply focused.

The arrows in Lead 3 of E and F point to a *notching* or slurring of R. In H, Lead 3 has a QRS group of the vibratory type (f).

B, F and H show a system of time lines produced by a different type of recorder. In these and all subsequent records like them, the time from the left of *each pair* of vertical lines to the left of the next pair is 0.20 second (see page 375).

CHAPTER II

THE NORMAL ELECTROCARDIOGRAM

THE electrocardiograms obtained from different normal hearts vary greatly from one another and yet any of these records may be called a normal electrocardiogram In order to define the normal electrocardiogram it is necessary then to describe the variations found in the different parts of the record as obtained from many normal hearts

It is difficult to give an exact description of the features of the normal electrocardiogram because there are often no exact limits to the variations of the normal but only a zone of gradual transition between normal and abnormal One can only say that certain features are usual in records from normal hearts that others are rare occurrences others very rare and still others have never been observed When very rare findings are considered we are always entitled to doubt that the heart is really a normal one perhaps some unsuspected otherwise hidden disease may have given rise to the features in question To say that the electrocardiogram is normal in the presence of rare features must always involve a certain *element of doubt* so that an attempt has been made whenever sufficiently detailed studies are available to indicate the degree of probability that a given feature is normal There are surprisingly few satisfactory detailed studies of records from supposedly normal hearts but the following statements *are based upon such as are available and upon such special* studies of diseased hearts as may give information about the normal electrocardiogram Two noteworthy contributions are Shipley and Hallaran s study of 100 normal males and 100 normal females and Barnes study of 50 normal males and 50 normal females References to their findings will be frequent

The differences between the analogous portions of the eight different records of Figure 11 are typical of those that appear

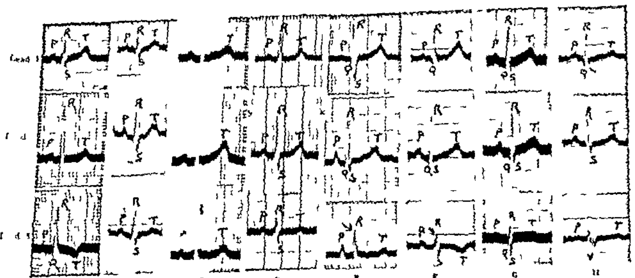


Fig. 11. Eight normal electrocardiograms to show the variation which may be encountered. In each case the upper electrocardiogram is by Lead 1, the middle one by Lead 2, and the lower by Lead 3.

Note the variations in the relative height of R in Leads 1, 2 and 3 as we pass from records A to H. Lead 2 always has the largest value. Lead 1 and Lead 3 vary reciprocally.

The fine vibrations of the line of the record, best seen in Lead 1 of B and E, are due to the electrical activity of the skeletal muscles of the extremities. The patient was not sufficiently relaxed. The blurring of the line of the record in B, F and H is due to the string shadow not being sharply formed.

The arrows in Lead 3 of E and F point to a notching or slurring of R. In H, Lead 3 has a QRS group of the vibratory type (?).

B, F and H show a system of time lines produced by a different type of recorder. In these and all subsequent records like them the time from the left of each pair of vertical lines to the left of the next pair is 0.20 second (see page 3?9).

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The differences between the analogous portions of the eight different records of Figure 11 are typical of those that appear

limits This knowledge has been accumulated slowly because of the difficulty of being certain that a given heart is surely normal Still by experience in correlating the electrocardiogram with the symptoms and physical signs in a large number of normal and abnormal individuals considering also their clinical progress and a certain amount of autopsy material it has become possible to make fairly definite statements as to the characteristics of the waves of a normal electrocardiogram When waves are found which do not agree with these standards we can predict that the contraction which produces them is in some way abnormal

The Normal P Wave

The normal P wave is a rounded elevation whose height lies between 0 7 mm and 2 5 mm in the lead which gives its largest excursion In the great majority of records 90 per cent according to Shipley and Hallaran the largest P will be in Lead 2 In the remaining 10 per cent they found the largest excursion divided about equally between Leads 1 and 3 Sometimes Lead 1 or Lead 3 may show a very small excursion or even none at all as in records A and E of Figure 11 In such records the other two leads will have an equal height Differences in the voltage of P in normal hearts probably depend in part upon the position and size of the auricles and their relation to the surrounding organs and in part upon variations in the physiological condition of the auricular myocardium

In about a third of all records from normal hearts the P wave shows a notching or doubling of its peak as in records A and E of Figure 11 This usually appears in Leads 2 and 3 but at times only in Lead 2 In Lead 1 it is less common but is still a normal finding A diphasic P wave (+ — or — +) having both an upward and downward deflection appears not infrequently in Lead 3 in records from normal hearts (1 per cent according to Ashman and Hull) but very rarely in Lead 2 and not at all in Lead 1 Notching of P should not be considered to indicate auricular abnormality A study by Lewis and Gilder of a group of 52 young men from whom possible abnormal individuals were most carefully excluded showed notching of P in one or more leads in 17

in records from different normal hearts They depend upon slight individual differences in the manner in which the contraction involves the muscle fibers of the auricles and ventricles The differences between different P waves and different QRS groups depend in part upon differences in the relative size and position of the muscle masses of various parts of the auricles or ventricles *and in part upon the position of the heart in the chest* The variations of P are probably less influenced by differences in the path of the contraction wave because of the diffuse radial spreading of the contraction through the auricular muscle from the sinus node The variations of the QRS group probably depend for the most part upon differences in the path of the contraction wave in the ventricles due to slight individual variations in the terminal ramifications of the auriculoventricular and Purkinje systems The variations of T in the three leads are due to variations in the regression of the electrical activity during the later phases of ventricular systole and early diastole

The height of the waves may vary in the same patient from time to time as the physiological condition of the myocardium varies but it is impossible to attribute slight differences in the height of waves which may be observed in records from different patients to differences in the physiological condition of the muscle This is because in different patients the actual electricity produced within the heart does not bear a constant relation to the amount obtained by leading from the extremities The available paths for short circuiting the current before it reaches the extremities must vary greatly in different patients and this will affect the amount of current reaching the extremities from which the leads are derived There is also the question of variations in the electrical resistance of the tissues about which we know but little The degree to which the margin of the lung is interposed between the heart and the anterior chest wall is certainly of considerable importance as can be readily seen by comparing the electrocardiogram of the same individual taken during inspiration and during expiration (Fig 18) During expiration the voltage will be found larger

Since the normal variations are so numerous and so marked it is necessary for the clinician to appreciate very clearly their

ally found so with rapid heart rates and in children Anything increasing the length of the path of the contraction would be expected to lengthen this time and it is also conceivable that a lowered functional condition (depression of conductivity) might prolong it

If the P wave is normal in all these respects—its height its direction in the three leads its form and its duration—we can conclude that the auricular contraction has passed in a normal way through a normal auricular muscle

In children the duration of P varies from 0 01 to 0 08 second the average duration being 0 06 second It is always upright in Lead 1 and rarely inverted in Lead 3 (1 per cent) or diphasic (2 per cent) The voltage of P has not been determined but the *maximum* value recorded was found to be 2 7 for P figures over 2 mm being rare The low limit of variation has not been studied except for the amplitude of individual leads Notching of P_1 and P was occasionally observed

The P R Level

The Auricular T Wave

From the end of the P wave until the beginning of the QRS group the line of the record rests near zero but rarely stays directly upon it in all three leads This deflection is due to a part of the auricular contraction process and since it is the auricular analogue of the T wave of the ventricular complex it has been called the auricular T wave It represents the summation of the T effects of the auricular fibers It is usually opposite to P in direction and comes to an end during the QRS group or shortly thereafter at a time when the voltage of T is beginning to develop Shipley and Halloran found this deflection in one or more leads in 95 per cent of their records and in Lead 2 in 90 per cent Its amplitude did not exceed 0 8 mm in their series In the author s normal series this deflection was downward in all three leads in 12 records and downward in two leads in 12 records The 2 remaining records showed no deflection in any lead It rarely is found to be slightly elevated above the zero level not over 0 01 mm There is at present no clinical signifi

records P was notched seventeen times P_3 ten times and
twice This agrees with the findings in a smaller group of norm
college students who were examined by the author In this grou
of 26 students P was notched in one or more leads nine tim
P_2 was notched seven times P_3 seven times and P_1 four tim
Ashman and Hull have *reported notching in 32 per cent of*
waves in their series of 100 normal individuals Shipley and H
loran in 30 per cent of their series It seems that notching mu
be accepted as one of the normal features of the P wave

The cause of notching of P is not known but it may be su
gested as a hypothesis that the normal P wave is composed
two overlapping electrical effects one of which is due to ea
auricle In the majority of instances these peaks fall so close
one another that no notching occurs Occasionally the path
the contraction in one auricle from the sinus node to the tip
the appendix or the base of the auricle may be longer than usu
so that the potential of that side will be slightly delayed in reach
ing its height Thus the peaks of the two electrical effects migh
not coincide as closely as usual and a notching of P would r
sult This hypothesis will help to understand some of the al
normal variations of P and will be mentioned further when the
are discussed

Duration of P The duration of the P wave the width of it
straddle measured on the base line is usually 0 08 second thoug
often as much as 0 10 second In 10 of the author s series of 2
normal students it was found to be between 0 08 and 0 10 second
In one case the duration was 0 06 second Shipley and Hallora
found the duration of P to be 0 12 second in 3 of their record
from 200 normal individuals and 0 11 second in 13 records Al
the others were between 0 08 second and 0 10 second Ashma
and Hull found the duration greater than 0 10 second in only
one of 100 records from normal individuals and in this record
it was 0 11 second It seems doubtful if a duration of 0 12 second
should be considered normal The duration of the P wave in
dicates the time necessary for the contraction to spread over the
whole of the auricular muscle and so must be reckoned from
the lead in which it is longest—usually Lead 2 We do not know
the factors which make this time unusually short but it is gener

cate the P R interval but the measurement from another lead should be used

The P R interval varies with the heart rate being shorter with more rapid rates and longer with slower ones It also tends to be shorter in children than in adults It may be as short as 0 12 second or as long as 0 20 second without being considered abnormal unless it should be found to be at or near this maximum in a child or in an adult with a heart rate of 90 or over In the examination of 1 865 supposedly normal young men Ferguson and O Connell found the P R interval longer than 0 20 second in 23 records longer than 0 21 second in 21 records longer than 0 22 second in 9 records Shipley and Hallaran did not find any case with P R longer than 0 19 second Ashman and Hull found but one case with P R as long as 0 20 second and none longer but they have emphasized the relation of heart rate to P R duration and the fact that a duration of 0 18 second with a rate above 100 is fully as significant as a duration of 0 21 second with a rate below 75 per minute The normal variations in the A V conduction time are dependent upon the functional condition of the conducting system short conduction time being dependent upon good functional condition and low grade of vagus activity while prolonged conduction may be due to a poor physiological condition of the bundle tissues or excessive activity of the vagus

In children the P R interval measures from 0 10 to 0 16 second the average being 0 13 second

The QRS Group

The QRS group is subject to an infinite number of variations because each of the three peaks can vary in height more or less independently of the others When examining this group of waves in the limb leads we must observe

1 The relative height and direction of the largest wave in each of the three leads

2 The presence and character of notching or slurring of the waves

3 The height of the largest deflection in any lead (voltage of QRS)

cance attached to this deflection though it is occasionally found in abnormal cases to be as much as 2.5 mm. It is usually large when the P wave is large. In the heart block records of Figures 51 B and 52 A it can be seen that the descending limb of P ends slightly below the zero level and the downward deflection remains for from *0.24 to 0.28 second*; so if this represents the usual duration of this deflection the time from the beginning of P to the ending of the auricular P wave would be from 0.31 to 0.38 second.

The P-R Interval

The interval between the beginning of the P wave and the beginning of the QRS group serves as a measure of the time taken by the contraction-producing impulse in passing from the sinus node to the ventricles. It measures the auriculoventricular conduction time and is called the P-R interval in spite of the fact that it is measured to the beginning of the QRS group irrespective of whether Q, R, or S is the first deflection of the group. This interval, as in Figure 10, is not usually of the same length in all three leads of the same record because one lead may be unfavorable for recording the beginning of the P wave or of the QRS group (Chapter IX). Since the interval is an index of the auriculoventricular conduction time it is obvious that the longest P-R interval which appears in any of the leads will afford the most correct measurement; this is found in Lead 2 in 75 per cent of normal records.

In following this procedure occasional records will be found in which the P-R interval is longer by 0.01 or 0.02 second than it would otherwise have been because of the inclusion in this interval of an initial isoelectric portion of QRS. This would have occurred in the measurement of P-R in Lead 1 of Figure 91. It would rarely be of clinical importance but occasionally an increase of 0.02 second produced in this way might be enough to make the P-R interval longer than normal. This situation may be suspected in clinical records if one lead shows a shorter QRS duration and a longer P-R interval than the others. In such records this long P-R measurement should not be taken to indi

tricular hypertrophy (Chapter *iii*) In normal hearts three factors combine in producing these variations of QRS and sometimes one sometimes another may be of predominant importance these factors are (1) The normal variations in the structure and distribution of the terminal arborizations of the auriculo ventricular bundle within the two ventricles (2) the position of the heart within the thorax whether transverse or vertical (3) the relation of the weight of the muscle masses of the right and left ventricles

Probably the most important cause of variations in the QRS groups of *normal* hearts is the variable distribution of the terminal arborizations of the Purkinje system within the ventricles Such variations would result in the stimulus arriving at various parts of the ventricular muscle in a slightly different order in different hearts so that the resulting production of electrical potentials would be different

Changes in the position of the heart upon the diaphragm result in gross variations in the direction of the long axis of the heart as well as in a rotation of the heart around its long axis In the transverse heart the rotation effect results in the anterior interventricular groove lying further from the left border of the heart so that the left ventricle is more visible anteriorly as seen in Figure 19 In the *vertical* heart the groove lies nearer to the left cardiac border so that the left ventricle is only slightly or not at all visible on the heart s anterior surface as seen in Figure 20 Each of these positions of the heart is apt to be associated with a special relation of the size of the peaks of QRS in the three leads This is especially so with the normal heart less so with hearts which have a preponderant hypertrophy of either ventricle The effect of a transverse position of the heart as it lies upon a high diaphragm is that R tends to be smallest in Lead 3 and records of the type of F G and H of Figure 11 or even of Figure 17 A may be obtained in normal hearts The effect of a vertical position of the heart—the so-called drop heart —is that the R wave is smallest in Lead 1 and records like A and B of Figure 11 are usual occasionally records like Figure 16 A are seen in normal hearts

It does not seem probable that the relative mass of the two

4 The duration of the QRS group is measured by its width on the base line

In all three leads of the usual normal record there will be an upward peak called R (Fig 11) It may be relatively small in Lead 1 or Lead 3 but it usually reaches a height in Lead 2 not exceeded in any other From such a series of records as Figure 11 we can see that as the height of R_1 diminishes in relation to R_2 so does the relative height of R_3 increase If one will follow the series of records F D C B A it will be seen that R is plainly larger than R_3 in record E while in record A these have become more nearly equal in size and R_1 is relatively small Likewise in the series C D E F G as the height of R_3 decreases in relation to R the relative height of R_1 increases R_1 and R_3 are seen to be reciprocal and in this we can discern the influence of Einthoven s law of the leads which is discussed in Chapter IX

Excursion Lead 1 + Excursion Lead 3 = Excursion Lead 2

The variations of the waves Q and S bear a certain relation to the relative heights of R_1 and R_3 Curves with relatively small R_1 tend to have a deeper S_1 than do curves with large R_1 and to have S_3 small or absent Q is usually largest in Lead 3 in such curves and absent in Lead 1 Conversely, those curves with relatively small R_3 tend to show the S wave better developed in Lead 3 than in Lead 1 while Q is better developed in Lead 1 than in Lead 3 These things will be apparent if the Q and S waves of records A B and C are compared with the same waves of records E F G and H

When R has a relatively small excursion in Lead 3 it is common for the QRS group in that lead to have *only* small excursions so that it is rather a vibratory complex as in records G and H Such vibratory complexes are rare in Lead 1 even when the value of R in that lead is relatively small When found they are not to be considered as normal There is considerable clinical interest in this division of the normal curves into three general groups as indicated (1) those with relatively small R_1 and large R_3 (2) those with R_1 and R_3 about equal and (3) those with relatively small R_3 and large R_1 The relative height of R_1 and R_3 will be seen later to have a bearing on the diagnosis of ven

will not be exceeded more than once in one hundred and once in one thousand observations Measurements of this sort may form a basis for the determination of normal and abnormal features of the electrocardiogram It seems that a finding which may be expected in less than 1 per cent of normal electrocardiograms should be regarded as probably abnormal and that one which may be expected in less than 0 1 per cent of normal electrocardiograms should be regarded as almost certainly abnormal

Large Q_3 It has been suggested elsewhere by the author that if the Q wave in Lead 3 exceeded 25 per cent of the largest peak of QRS found in any of the limb leads such a Q wave was to be considered abnormally large It was emphasized that the downward deflection must be followed by an R in order to be called Q and must not be preceded by any upward deflection It was also indicated that a Q_3 of such value would be considered normal in the presence of right axis deviation of QRS (see page 137)

Occasionally records obtained from normal hearts will show a Q wave in Lead 3 larger than the limits just stated Because such records are more frequently found in hearts which are diseased it is important to call attention to the possibility that a large Q_3 may occasionally be a normal finding Shipley and Hallaran found four such records in their series all in males a frequency of 2 per cent Three of these records showed an inverted T_3 and the fourth a diphasic T_3 Other observers have not found a large Q_3 so frequently in normal individuals The author found only two such records in a collection of 277 normal persons from various sources and Edeiken and Wolferth found only one in records from 826 normal college students The calculated incidence of an abnormally large Q_3 in the 1 303 normal young adults of these two series is approximately 0 55 per cent Most observers who have noted this condition in normal hearts have found that it appears most frequently though not exclusively in individuals with a broad type of chest It has also been found in 5 per cent of records from the hearts of normal women during pregnancy and has disappeared from the record of these same women after delivery It is not uncommon in the electrocardiograms of normal infants and young children In normal hearts it appears to be due to a displacement of the apex upward

ventricles has much influence upon the QRS group in a heart of normal size but if so it would probably be analogous to that found with ventricular hypertrophy (Chapter III)

Wilson has observed that in all of the standard limb leads and in precordial leads as well large Q waves tend to be associated with large R waves and large S waves with small R waves so that the reverse association large Q with small R and large S with large R should be viewed with suspicion as possibly being abnormal He has also obtained from a series of electrocardiograms of 104 normal men between the ages of 20 and 30 measurements of the height of the different waves which are shown in Table I This table which is condensed from the original also shows a figure for the maximum value of these waves which

TABLE I

MEASUREMENTS OF 104 NORMAL ELECTROCARDIOGRAMS

	MEAN	STANDARD DEVIATION	MIN	MAX	1 PER CENT	0.1 PER CENT
	mm	*mm*	*mm*	*mm*		
P_1	0.50	0.21	0	1.2	1.12	1.42
P_2	1.15	0.40	0.3	2.5	2.25	2.73
P_3	0.77	0.39	0.2	1.8	1.81	2.09
Q_1	0.36	0.45	0	2.0	1.75	2.46
Q_2	0.58	0.59	0	2.5	2.36	3.25
Q_3	0.61	0.66	0	3.0	2.64	3.69
R_1	5.73	2.74	1.5	19.4	14.20	18.53
R_2	11.90	3.96	4.0	23.6	22.28	26.43
R_3	7.99	4.41	1.0	20.0	19.54	24.17
S_1	1.78	1.27	0	6.0	5.29	6.81
S_2	1.83	1.52	0	8.0	6.42	8.72
S_3	1.39	1.67	0	13.0	6.55	9.19
T_1	2.05	0.97	−0.5	5.5	{4.92, 0.44}	6.31
T_2	2.99	1.37	0	8.0	{6.47, 0.10}	7.81
T_3	1.22	1.22	−2.0	5.5	{4.31, −1.34}	5.51

This table gives the mean the standard deviation and the maximum and minimum measurement of the waves of the electrocardiograms of 104 normal men between 20 and 30 years of age In addition to this the last two columns give the measurement which the mathematical probabilities indicate will not be exceeded more often than once in 100 times (1 per cent) or once in 1000 times (0.1 per cent) The measurements are expressed in tenths of a millivolt which in the ordinary electrocardiogram equals 1 mm
[illegible] two values of the variable [illegible] than [illegible] first

possibly accompanied by a counterclockwise rotation of the heart about its long axis

Kossmann has studied the Q wave in records from 178 normal young people and has found the maximum normal values of 2 mm in Lead 1 2 5 mm in Lead 2 and 3 mm in Lead 3 The maximum normal Q expressed as a percentage of the highest QRS peak in the particular lead in which it occurred was 15 per cent for Q 1 20 per cent for Q 2 and 25 per cent for Q 3 These maxima are only to be applied to records in which there is not an R wave of 19 mm or more and only to Leads 2 and 3 when there is not right axis deviation He found differences between records obtained in the sitting and in the recumbent positions but even so the above limits hold good for either position With the patient in the sitting position Q_3 was unaffected by deep inspiration in 40 per cent of the cases expiration increased the size of Q_3 in 50 per cent and decreased it in 10 per cent He did not find a single Q 3 greater than 25 per cent of the largest peak of QRS in the limb leads

One occasionally encounters records in which it is difficult to decide whether or not the downward deflection of the QRS group should be called Q This doubt arises under three circumstances (1) When as in Figure 12 A the Q wave is preceded by an upward deflection which as it increases and decreases with the respiratory variations in the position of the heart is sometimes present and sometimes absent In those complexes which show this small upward deflection the complex is of the M type and the downward deflection should not be called Q whereas in those where there is no initial upward deflection it is quite properly so called This condition must be recognized as a borderline one and must be decided arbitrarily If the downward wave is not usually preceded by an upward deflection that is if Q is more frequent than the M complex the record may be considered as having the usual significance attached to the presence of Q in the particular case If however the wave is usually preceded by a small upward deflection if the M complex is more frequent then the record should be considered as having the usual significance attached to the presence of an M complex

(2) The respiratory variation of the waves of Lead 3 may

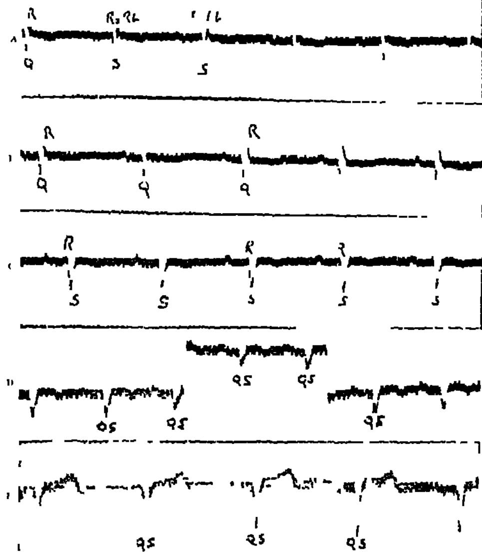


Fig. [illegible] Records to illustrate situations encountered in the naming of the Q wave. These records are all by Lead 3 but complexes similar to some of these are occasionally encountered in Lead 1 and in precordial leads.

A The first cycle shows a Q followed by a notched R. After this the complexes are of the M type with a very small initial upward deflection *Ra* and a larger secondary upward deflection *Rb*. In such a record all downward deflections should be similarly named, in this case S.

B illustrates variations in the size of Q. The two smallest Q waves were less than 25 per cent of the highest R of this patient's electrocardiogram but because large Q waves were more frequent the record should be considered to show this feature. The record also shows variations in the height of R, this peak being scarcely noticeable in the second cycle.

C A downward deflection *S* preceded by a tiny and inconstant upward deflection *R*. The cycles with an R are more frequent so the downward wave is called S instead of QS.

D and E illustrate QRS groups without an R wave either before or after the downward deflection. These downward waves are called QS.

possibly accompanied by a counterclockwise rotation of the heart about its long axis

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(2) The respiratory variation of the waves of Lead 3 may

result in a record showing Q waves of varying height sometimes smaller and sometimes larger than 25 per cent of the maximum deflection of the QRS group as in Figure 12 B A change in the position of the patient by changing the position of the diaphragm is also capable of changing the size of Q_3 It seems better to consider that a large Q_3 is present when the majority of the Q waves of the record reach the required size rather than to attempt to decide upon any other grounds A deep inspiration in such a patient will usually diminish the size of Q_3 but the fact that it does so is not surprising in view of our knowledge of the changes in the position of the heart with inspiration nor should diminution or disappearance of Q_3 on inspiration have any bearing upon whether or not the Q wave in question is to be considered normal or abnormal

(3) When the QRS groups *show no upward deflection* the *sole deflection being a downward one* * the author believes that it is better at present to call this deflection QS rather than Q or S as has been previously recommended The reasons for this *are fully discussed in Chapter IX to which the reader is referred*

Notching and slurring of the QRS group Notching is the appearance of two minor peaks pointing in the same direction without the intervening oppositely directed apex crossing the zero level It is due to an actual double change in the direction of the motion of the string shadow It is seen in R_3 of records D F and H of Figure 11 and in Figure 10 Leads 1 and 2 Slurring is a thickening or broadening of a portion of the line producing QRS and is due to a transient retardation of the movement of *the string It occurs normally (1) at the peak of the Q R or S* waves as in R_2 of Figure 11 A and B (2) when the transition to or from the zero level is gradual rather than abrupt as in the descending limb of R_3 in records A and C and in the ascending limb of R_1 in record A (3) when the total amplitude of a peak is small as S of all three leads of Figure 11 B in Q_3 of Figure 11 A and Q_1 of Figure 11 H The peak of R of Figure 10 shows an unusual degree of slurring

Notching of the R wave is *especially prone to occur in Lead 3 when R is relatively small in this lead and R_1 is about equal to*

* Such records are not obtained from normal hearts

R (Figs 11 F and H) The QRS of Lead 3 in these records is often composed of a series of small vibrations as in record 11 so that it is difficult or impossible to name the individual peaks Such a QRS group may be spoken of as vibratory or splintered * Notching or slurring of QRS is occasionally seen in Lead 1 when the excursions are relatively small in this lead and R and R_3 about equal in size but it is rare to see anything like the vibratory or splintered wave groups which are so common in Lead 3 If they are found in Lead 1 the record cannot be considered a normal one

If the process which causes notching is less in degree and occurs during the upward or downward limb of a large wave it is as though the notch were stretched out and it may be changed to a slurring or thickening of a section of one limb of R or S as in Lead 3 of Figure 11 E This has the same significance as notching It has a different sort of origin from the slurring so frequently seen as the R or S waves leave or approach the base line

When notching occurs at the peak of a wave it results in a broadening or thickening of the peak as in Lead 2 of Figure 10 which sometimes may amount to a true notch as in Figure 11 F Lead 3 The normal thickening of the line as it changes direction at the peak of R or S must be distinguished as different from such notching or slurring

Notching or slurring of the waves of the QRS group can be considered a normal phenomenon when it is found at the beginning or end of the group or if in the course of one of the ... t ... anne ...

... normal only when it occurs at the beginning or the end of the QRS group and very near to the base line Shipley and Hallaran however observed notching or slur

* On reviewing a normal series of 18 persons 52 recorded by Lewis and ... by the author it was found that whereas T was turned downward in 1? records there were 13 of these which also had notching or splintering of the QRS group in Lead 3 Moreover of the 3? cases with notching or splintering of QRS a downward T was found in 13 (40 per cent) a frequency about double that of a downward T in the series as a whole i.e. 1? times in 8 cases or 22 per cent It seems evident that whatever feature of the normal electrocardiogram leads to a notching of QRS in Lead 3 tends also to produce a downward T wave in this lead

ring remote from the base line in both Leads 2 and 3 in three of their 200 supposed normals The rarity of this occurrence is such that a record showing it must at least be viewed with suspicion It is probably not normal to find notching near the peak of R in a lead of relatively large excursion

Notching and slurring are such evident features of abnormal electrocardiograms that it is necessary to realize that they may sometimes be a normal feature Their basis in the changing potential produced during ventricular contraction is discussed in Chapter IX and also what this may mean when referred to muscle function

The voltage of QRS It has been stated that the voltage of a wave is approximately measured by the size of the deflection in the lead giving this wave its largest value In Figure 11 then the voltage of the QRS group would be best shown by R in all records but II and in this record by both R_1 and R In this figure the value for QRS varies from 17 mm to 8 mm in different records

In Lewis series of 52 normal students the variations in the height of the largest wave of QRS were from 5 5 mm to 16 5 mm with an average of 11 mm In my own normal series the height varied from 8 mm to 23 5 mm but these were college students and engaged in a certain amount of competitive athletics while Lewis series were medical students who presumably were older and not so much concerned with athletics

Shipley and Hallaran found three women with a voltage of 5 mm or less and three men with a voltage of between 7 mm and 5 mm The maximum excursion in their series was 19 7 mm It is probable that the figures at either extreme might not be normal and so from a study of the frequency of occurrence of the different values in the combined series of 278 normal persons it seems proper to set a minimum value of 5 mm and a maximum value of 20 mm as the normal limits for the height of QRS in its largest lead but not proper to consider these limits as an exact border line between normal and abnormal The average for the combined series was 12 mm Shipley and Hallaran found the average value for males to be 12 1 mm and for females 10 8 mm

Whenever one lead gives QRS a very small relative value and

R is equal in the other two leads the highest recorded value should properly be increased by 15 per cent to obtain a truer measure of its voltage.* In such records then the normal limits would be from 4.4 mm to 17.4 mm. The mathematical reasons for this are evident in the explanation of Einthoven's table (page 110).

Variations in the voltage of QRS occur from time to time in normal persons and frequently in patients with cardiac failure. Provided that the QRS group does not change its form in any other way, variations in its voltage can usually be shown to be coincident with variations in the nutritional state of the heart muscle. Small values seem to mean a muscle which is below par, perhaps through lack of exercise or perhaps through fatigue or some other fundamental disturbances in its nutrition, while large values mean a strong well-nourished muscle. During convalescence from an acute disease, for instance, the value of QRS increases, as it also does when the condition of the circulation improves after cardiac failure. The differences noted between the voltage of QRS in the two series of normals just mentioned may be explained on this basis, the more sedentary group of Lewis showing smaller values than the more athletic group of the author.

The differences in the voltage of QRS which are found in the records of different normal individuals are largely due to variations in the order in which the different portions of the ventricular muscle receive their stimulus through the ramifications of the auriculoventricular system. They are also largely affected by the position of the heart within the chest, particularly by its contact with the anterior chest wall. There tend to be large excursions if the contact is extensive and small excursions if the

* The value of the largest peak of QRS recorded in any lead differs from the manifest value of QRS more and more as the direction of the potential produc[illegible] is more nearly perpendicular to the line of any lead. The recorded value [illegible] 8 [illegible] per cent of the manifest value when the direction of the [illegible] exactly perpendicular to a lead. An approximately perpendicular [illegible] for the QRS group may be recognized from the three leads of the record by the fact that in one lead there is a very small relative value [illegible] the [illegible] leads the values are [illegible]

[illegible] value of the

heart is largely separated from the chest wall by overlapping pulmonary or mediastinal tissue The short circuiting of the heart currents by the viscera is an important factor and largely unpredictable in determining the relation of the amount of current within the heart to the amount recorded in leads from the extremities in different individuals

Duration of QRS Normal hearts show differences in the duration of QRS The time from the beginning of Q or of R whichever is the first to develop to the end of R or S whichever finishes the group is the time consumed by the spreading of the contraction throughout the ventricles in other words the time from the first ventricular activity until the contraction has come to involve the whole ventricular musculature The longest time measurable in any one of the three leads gives its correct measurement

It must be emphasized that the duration of QRS must be very carefully measured as error may arise because of uncertainty as to the exact point at which the QRS deflection begins and ends Several complexes in each lead should be carefully inspected as small deflections at the beginning or ending of QRS may vary from one cycle to another and when present may add 0 01 second or occasionally 0 02 second to the measurement of the duration of QRS These small deflections are obviously a part of QRS but they are missing in certain cycles because the respiratory movement of the heart makes their potential perpendicular to the line of the lead during these cycles so that they are not recorded

In the normal hearts of adults the duration of QRS is found to vary between 0 06 second and 0 10 second Shipley and Hallaran found it to be 0 11 second in two records and 0 12 second in one of their normal series of 200 McGinn and White found three tracings with the duration of QRS greater than 0 10 second none greater than 0 11 second They also noted a correlation between the duration of QRS and the heart size as indicated by the height of the individual A longer duration for QRS was found in those with greater height and a shorter duration in shorter individuals McGinn and White found QRS to have its longest duration in Lead 3 in 44 of their 100 normal individuals in Lead 2 in 37 cases and in Lead 1 in 19 cases so that it is evidently unreliable to always depend upon the measurement of the QRS duration

of any one lead chosen arbitrarily Ashman and Hull found a variation from 0 06 second to 0 095 second in their series of 100 normal individuals They also found a correlation between small size and short QRS duration and large size and large QRS duration Luderitz has measured the QRS duration in 500 records from normal hearts and found the frequency of the various durations of QRS as shown in Table II It will be seen that Lead 2 and Lead 3 tend to give a longer measurement than Lead 1 and that a duration of 0 12 second is very rare

TABLE II

DURATION OF QRS (From Luderitz)

DURATION OF QRS	LEAD 1	LEAD 2	LEAD 3
Second	Per Cent	Per Cent	Per Cent
06	3	0 8	1 8
07	26	19 0	17 0
08	46	43 0	43 0
09	15	25 0	21 0
10	4	10 0	10 0
11	1	1 0	1 8
12	0	0 8	0 4

The duration of QRS is also somewhat influenced by *the heart rate* Ashman and Hull and Luderitz have both emphasized this the duration being shorter with more rapid rates and longer with slower rates The fact that children's hearts always have a very brief QRS suggests that a short auriculoventricular conduction system combined with a short path through the ventricular muscle will give a brief QRS group It is also probable that variations in the functional condition of the Purkinje tissue or perhaps even of the ventricular muscle may shorten or lengthen the duration of QRS

The QRS group in children The QRS duration is shorter in children than in adults measuring from 0 05 to 0 09 second the latter measurement being found in only 2 cases in a series of 100 normal children The average was 0 065 second According to one group of authors the average was found to increase gradually with

increasing age according to another author it was not influenced by age

The voltage of QRS cannot be stated for the authors who have investigated children's electrocardiograms have confined themselves to measuring the height of the individual waves in the various leads The average R_2 is higher than found in normal adults and as most of the records did not show right or left axis deviation it is probable that this is the best available indication of the voltage of QRS The averages as found by Lincoln and Nicholson vary from 11 to 16 mm in different age groups with variations from 6 5 to 32 mm the usual low range being 7 mm the usual high range being 26 mm Hafkesbring Drawe and Asher however found R_2 averaged 11 5 mm the range being from 5 to 22 mm

The direction of the electrical axis ascertained by the method of Dieuaide has been found by Hafkesbring and his associates to vary between 20° and 110° in a series of 100 normal children with the exception of 3 cases which gave values outside of these limits Lincoln and Nicholson found in their group of 222 cases 3 children showing a definite predominance of the right ventricle and 8 showing predominance of the left ventricle according to Einthoven's signs

THE S T SEGMENT

The beginning of the first limb of T may be at the zero level or slightly above or below it It often will follow the zero level for a few hundredths of a second before starting to curve upward or downward toward the peak of T lying either at zero as in Lead 2 of Figure 11 A or slightly above or below it In other records and usually in other leads of the same record the T wave starts immediately to develop its first limb without any such approximately isoelectric portion (Leads 1 and 3 of Fig 11 A) The first portion of the T wave has been called the R T or S T interval or segment and this terminology has led to some confusion of nomenclature by leading to an erroneous consideration of this portion of the T wave as a separate entity The term segment seems to be less liable to lead to this inaccuracy of thought

and so is preferable. It must be emphasized that the S-T segment is merely the first limb of the T wave and ends at the peak of T. Figure 13 illustrates some of the types of S-T segment com-

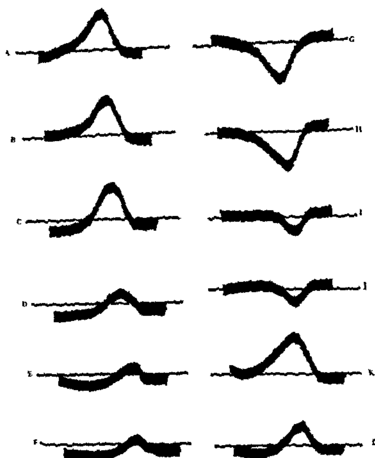


Fig. 13. The S-T segment. *It will be noted that in* A, E, G *and* L *the S-T segment starts at the zero level; in* B *and* K *above this level; and in* C, D, F, H, I *and* J *below it. The zero line is drawn through the top of the string shadow when the T wave is upward and through the bottom of the string shadow when the T wave is downward. Types* F, E, G, H, I *and* J *would not be normal if found in Leads 1 and 2 but would be normal in Lead 3.*

monly *encountered* in normal records. The first two types A and B are those usually found in *Leads 1 and 2*; types C and K are occasionally found in these leads, but any type shown in the figure may be found in *Lead 3*. This figure does not include all of

the normal forms taken by the S T segment and the T wave but the varieties not shown are rare It will be observed that the S T segment tends to be convex toward the isoelectric level When the peak of T is upward, the S T segment is concave upward as in A B C D E F, K and L It may start slightly above the zero level as in B and K or at this level or slightly below it as in C D and F and pass to an upward T by the usual upward concavity When the peak of T is downward S T will be concave downward as in G H and J or may be straight as in I starting either at or below the zero level In normal records S T will occasionally be found to begin above the zero level and then curve downward to form an inverted T and in Lead 3 it may occasionally, as in J originate below zero and curve slightly upward before turning downward thus simulating the coronary T wave (Fig 66)

When the S T segment starts below zero and curves upward from its point of origin to the peak of T as in C and D of Figure 13 the resulting T wave should not be referred to as diphasic The term diphasic T had best be restricted to those curves in which the S T segment moves in one direction away from zero forming an apex and then returns to zero and crosses it forming a second apex so that the peak of T is found in the opposite direction to the first apex as seen in curves E F and L of the figure which represents the —+ type of diphasic T wave Curves J and K should not be referred to as diphasic because although the apices are in opposite directions the peaks are on the same side of the zero level Diphasic T waves occasionally occur in normal records in Lead 3 rarely in Lead 2 and never in Lead 1 Attention is again called to the suggestion made on page 361 that the term diphasic should be followed either by the signs —+ or by the signs +— to indicate the direction of the first and second apices

The S T segment cannot be considered as having any definite point of ending before the peak of T but is merely the first limb of the T wave One of its most important features is the level of its beginning the point of origin of the T wave from the QRS group which may be called the S T junction Deflection of this S T junction above or below the zero level of the record is in part due to the auricular T wave which may still persist at this time of the heart cycle and in part to the potential produced by

the ventricular muscle at this time The auricular T wave is responsible for the deviation of the string shadow between the end of I and the beginning of QRS but we know that this deflection does *not* always remain constant from the time it disappears into QRS until its ending during the first limb of the T wave Usually however its amplitude is so small as to be negligible

The deflection at the S T junction was found in the author's series to be usually above the zero level of the record in two or in three leads (16 cases) occasionally below it in one or more leads (7 cases) and in 3 cases exactly upon it

Shipley and Halloran found that in 200 complexes without auricular T wave the S T level (junction) was usually above zero in Lead 1 always above in Lead 2 and about equally divided in Lead 3 between a position above and below zero There was an interesting difference between the sexes in Leads 1 and 3 males having this level more frequently above zero in Lead 1 and

TABLE III

LEVEL OF THE S-T JUNCTION

		Number with Elevation		Maximum Elevation (mm)		Average Elevation (mm)	Number with Depression		Maximum Depression (mm)		Average Depression (mm)
		B	S and H	B	S and H	B	B	S and H	B	S and H	B
Lead 1	Males	33	34	0 9	0 7	0 3	3	3	0 3	0 2	0 1
	Females	25	32	0 8	0 3	0 2	8	4	0 2	0 4	0 1
Lead 2	Males	41	69	1 0	1 3	0 5	6	0	1 0	—	0 6
	Females	17	45	0 5	0 5	0 3	18	5	0 8	0 3	0 3
Lead 3	Males	32	23	0 8	0 5	0 3	11	3	0 6	0 3	0 2
	Females	15	13	0 4	0 2	0 2	18	7	1 0	0 5	0 2

Measurements of the level of the S-T junction in relation to the P R level in normals B = 50 males and 50 females reported by Barnes S and H = 100 males and 100 females reported by Shipley and Halloran.

the normal forms taken by the S T segment and the T wave but the varieties not shown are rare It will be observed that the S T segment tends to be convex toward the isoelectric level When the peak of T is upward the S T segment is concave upward as in A B C D E F K and L It may start slightly above the zero level as in B and K or at this level or slightly below it as in C D and F and pass to an upward T by the usual upward concavity When the peak of T is downward S T will be concave downward as in G, H and J or may be straight as in I starting either at or below the zero level In normal records S T will occasionally be found to begin above the zero level and then curve downward to form an inverted T and in Lead 3 it may occasionally as in J originate below zero and curve slightly upward before turning downward thus simulating the coronary T wave (Fig 66)

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females having it more frequently below zero in Lead 3 Their measurement of the S T junction in relation to the P R level gave values as shown in Table III which also includes analogous measurements by Barnes Figure 14 A and B show two records from normal young men with unusually large deflection of the S-T junction Shipley and Hallaran observed a direct relation between the size of the deflection of the S T junction and the size of the QRS deflection in the same lead so that they did not find their maximal values with a QRS group of low amplitude They found 20 records with downward T_3 and upward convexity of the S T segment similar to the coronary T wave There were also 10 records with upward T and a downward convexity of the S T segment

The S T segment may remain at the level of the S T junction for a few hundredths of a second perhaps for as long as 0 12 second or 0 16 second but usually starts at once or almost immediately to move away from zero toward the peak of the T wave This part of the T wave is of great importance because of the significance of the variations occurring as a result of myocardial disease and under the influence of digitalis It will sometimes vary when the T wave does not or it may vary more definitely than does T

In children the S T junction is very rarely deviated from the isoelectric level Hafkesbring and his associates found it slightly above zero in Lead 2 in 7 cases in their series once in Lead 1 and once in Lead 3 The size of the deflection was usually 0 5 mm never as much as 1 mm In one case there was a slight downward deflection (0 75 mm in Lead 2)

THE T WAVE

The T wave was formerly considered the last of the ventricular waves but it now seems that U must also be included T has several special features which should be noticed (1) its direction in the three leads whether upward or downward (2) its maximum excursion in whichever lead this occurs (voltage) (3) the form of its initial limb (4) the form of its apex (5) its duration

Direction of the T wave In records from normal hearts the T wave is directed upward in Leads 1 and 2 though the size



Fig. 14 Records with unusually large deviation of the S T junction (A and B) and with inverted T 2 (C) occurring in otherwise normal young men

A and B Record A was obtained from a man who was a member of his college track team In this record the S T junction is + 0 6 mm in Lead 1 1 2 mm in Lead 2 and 0 8 mm in Lead 3 In B the values are 0 3 mm 1 0 mm and 0 [illegible] mm respectively

C and D These records were taken with the patient in the seated (C) and in the dorsal (D) positions It will be seen that the T wave which is diphasic (+ −) or inverted in Lead 2 and inverted in Lead 3 when he is erect becomes upright in Lead 2 when he is lying down

or even inverted T_3 just as it tends to produce a small or inverted QRS group in Lead 3. Shipley and Hallaran found 21 records with left axis deviation of QRS among their normals (10 per cent) and [illegible] of these had an inverted T_3. In the combined normal series of Lewis and of the author there were seven records (10 per cent) that had a small notched QRS_3 similar to that of Figure 11 F with R_1 equal to or larger than R_2 so that the electrical axis was almost if not quite to the left of normal. All of these records also showed a downward T_3. On the other hand a person with a long narrow chest because of the resulting vertical position of the heart (page 89) would tend to have T relatively small in Lead 1.

As with the QRS group however the position of the heart is only one cause of variation in the direction and height of the T waves in the three leads. Variations in the spreading of the contraction wave will lead to variations in its recession so that certain features of QRS are apt to be associated with certain directions of the T wave in the three leads. The reader is referred to Chapter IX for a discussion of this subject. The changes in the T wave which are often associated with an abnormal direction of the electrical axis of QRS (page 99) suggest that an inverted T_3 may sometimes result from the influence of a predominance of the right ventricle during contraction. In normal hearts without right axis deviation of QRS the inversion of T_3 may be related to an unusually great influence of the right ventricle during the latter part of the contraction. In line with this idea is the observation that in the combined normal series of Lewis and of the author there were sixteen records with downward T_3 (20 per cent). Seven of these have been mentioned above as showing a tendency to or a slight left axis deviation of QRS and the inverted T_3 in these may have been due to an upward tilting of the cardiac apex. The other nine showed a QRS in Lead 3 like that of Figure 11 A with a Q a large R and no S wave which is like the QRS of Lead 3 with right axis deviation (Figure 16 B C D). Five of these nine records showed another feature suggesting right axis deviation of QRS that is a relatively small R_1 with well marked S_1 so that S approached R in size if it did not equal or

in one of these leads may be small in relation to the maximum shown in the other. About one third of the records from normal hearts have a downward T in Lead 3, as in Figure 11 A and F. About one seventh show a diphasic (−+) T wave in Lead 3, as seen in Figure 76 A, which is a record from a normal heart, though often the phases are not so marked as in this record. The remaining two thirds will show an upward T_3. Shipley and Hallaran found two records in their series with notched T_2 and one with a diphasic T_2.

There is a small number of individuals whose hearts apparently are normal but who nevertheless have an electrocardiogram with an inverted T wave in Leads 2 and 3 if the record is obtained with the patient seated erect. These individuals are usually of the hyposthenic type and when they assume the reclining position the T wave is found to be upright in Lead 2 and possibly also in Lead 3. Records of such an individual are seen in Figure 14 C and D. The wearing of an abdominal supporting belt usually will return the T wave to normal in Lead 2. These individuals sometimes show the symptoms described as neurocirculatory asthenia; sometimes they are apparently quite normal. The reason for this change of the T wave with position is not understood. It has been suggested that it is due to the changed contact of the heart with the chest wall and diaphragm; it has also been ascribed to a changed blood flow in the coronary sinus resulting from the change in position. It is important to bear in mind that when records are taken with the patient seated an inversion of T_2 and T_3 may be found rarely in a normal heart.

The direction and form of T in the three leads depends upon the direction of the current within the heart producing the wave and upon the relation of this current to the three leads. One could determine the electrical axis of T more easily than that of QRS because its peak is more nearly in phase in the three leads, but it is not customary to do so, as a description of the direction of the peak in each lead is simpler and gives the same information.

A downward T_3 may at times be due to a high position of the diaphragm. This would tip the apex of the heart upward and rotate it slightly, and tend to produce a relatively small

or even inverted T_3 just as it tends to produce a small or inverted QRS group in Lead 3 Shipley and Hallaran found 23 records with left axis deviation of QRS among their normals (10 per cent) and 15 of these had an inverted T_3 In the combined normal series of Lewis and of the author there were seven records (10 per cent) that had a small notched QRS_3 similar to that of Figure 11 F with R_1 equal to or larger than R so that the electrical axis was almost if not quite to the left of normal All of these records also showed a downward T_3 On the other hand a person with a long narrow chest because of the resulting vertical position of the heart (page 89) would tend to have T relatively small in Lead 1

As with the QRS group however the position of the heart is *only one* cause of variation in the direction and height of the T waves in the three leads Variations in the *spreading of the* contraction wave will lead to variations in its recession so that certain features of QRS are apt to be associated with *certain directions* of the T wave in the three leads The *reader is* referred to Chapter IX for a discussion of this subject The *changes* in the T wave which are often *associated with an abnormal* direction of the electrical axis of QRS *(page 99) suggest that an inverted* T_3 may sometimes result from the influence of a predominance of the right ventricle during *contraction In normal* hearts without right axis deviation of QRS *the inversion of* T_3 may be related *to an* unusually *great influence of* the right ventricle during the latter *part of the contraction* In line with this idea is the observation *that in the* combined normal series of Lewis and of the author there were sixteen records with *downward* T_3 *(20 per cent)* Seven of these have been mentioned above as showing a *tendency* to or a slight left axis deviation *of QRS* and the inverted T_3 in these may have been due to an upward *tilting of the* cardiac apex The other nine showed a QRS *in Lead 3* like that of Figure 11 A with a Q a large R *and* no S wave *which* is like the QRS of Lead 3 with right axis deviation *(Figure 16* B C D) Five of these nine records showed another feature suggesting right axis deviation of *QRS that* is a relatively *small* R_1 with well marked S_1 so that S approached R in size if it did not equal or

exceed it Such records as these nine suggesting the predominant effect of the right ventricle upon T may result from a disproportionately short path along the right branch of the auriculoventricular bundle or an unusually long path in the left branch

Voltage of T If the height of T be measured in the lead showing its largest excursion, it will rarely be found greater than 6 mm in records from normal hearts and rarely less than 1 mm The variable relation of this measurement to the voltage of T must be borne in mind (page 410) Shipley and Hallaran found the largest excursion of T to lie between 1 5 mm and 5 mm in all but 12 of their series * Four gave measurements of 1 1 mm one 1 mm and one 0 8 mm Six records all from men gave measurements over 5 mm three of these between 5 mm and 6 mm and three over 6 mm the largest being 6 8 mm The combined series of Lewis and the author showed the largest excursion of T between 1 5 and 5 mm in all but eight records five of these showing a value of 1 mm and three a value of 5 5 mm In these three series of 275 records six records gave a value of 1 mm one of 0 8 mm nine gave a value more than 5 mm and four of these between 6 mm and 6 8 mm (1 per cent) Only 2 per cent then gave a value of less than 1 mm or more than 6 mm for the largest excursion of the T wave From a consideration of the frequencies of the various measurements it would seem that records with the largest excursion of T less than 1 mm probably are not to be considered normal nor are records with the largest excursion greater than 6 5 mm

As with the QRS group (page 49) this measurement records only 87 per cent of the voltage when the T wave of one lead is practically isoelectric and the other two approximately of equal size This was the case in all of the records of Lewis series which gave a 1 mm measurement for the largest T wave If the limits of normal height are between 6 5 mm and 1 mm then in such records the limits would be reduced to 5 mm and 0 8 mm

The physiological causes of variations in the voltage of T are

* Personal communication

not known In a general way it seems to be influenced by the strength of the cardiac contraction being large in the hearts of athletes and in the same person becoming larger after exercise and smaller after a period of acute illness That the voltage of T may be affected by normal physiological influences has been proved by the demonstration of its decrease after meals in 7 of 9 normal young men The variations in the voltage of T do not parallel those of the QRS group exactly for an R wave toward the upper limit of normal may be seen in a normal person who has an average T wave and vice versa There is however a general parallel between the voltage of QRS and of T in normal records so that a large voltage of QRS is usually associated with a large voltage of T while the same is true as to low voltage A record should be viewed with suspicion which has a QRS voltage toward the upper limit and the T wave toward the lower limit or vice versa

It must be emphasized again that what has been said about the voltage of the QRS and the T waves is applicable only to records which arise by a normal process of contraction Abnormal modes of contraction in themselves change the voltage of these waves as is pointed out in Chapter II so that if for example the QRS group is notched or slurred the voltage of QRS and of T is likely for this reason to be different from what it would have been without the notching

The T wave in children is always directed upward in Leads 1 and 2 and as with adults is directed downward in Lead 3 in about one third of the cases it is diphasic in Lead 3 in about 7 per cent more The voltage of the T wave is somewhat larger on the average in children than in adults but from the measurements which have been published it is not clear what the maximum and minimum values might be The measurements of T have been made in each of the three standard leads separately without considering the relative heights in the three leads It seems from the available measurements of Lead 1 and Lead 2 that the maximum voltage may reach 9 mm but it is not possible to determine what the lower limit of the voltage of T might be owing to the way the statistics have been collected

Q T Duration

The duration of T cannot be considered to give an indication of the duration of the ventricular systole because of the variations which may occur in the duration of QRS It is best to consider the total duration of the ventricular complex (Q T) as a guide to the duration of systole From the very beginning *of the QRS group there are more and more muscle fibers en*tering into contraction until at its end all are contracting Even the intraventricular pressure begins to rise before the end of the QRS group is reached (Fig 8) so that it is quite illogical to consider the duration of T as indicating the duration of systole It is true that the total duration of the ventricular complex is greater than that of the mechanical contraction but these probably bear a more constant relationship than do the duration of T and the mechanical effect

The Q T duration must be determined from the lead in which it is longest Shipley and Hallaran found this to be Lead 2 in 95 *per cent of their series* Other things being equal the Q T duration will depend upon the heart rate In the author's series of normal individuals the heart rates and duration of the ventricular complex appeared as follows

Table IV

DURATION OF VENTRICULAR COMPLEX

Rate	Number of Cases	Duration (Seconds)		
		Average	Low	High
52	1	46		
60 to 69	8	40	36	42
70 to 79	10	375	34	40
80 to 89	4	355	34	36
90 to 99	3	34	34	34

This series is too small to enable us to draw far reaching con*clusions but it appears that if with a heart rate* of 70 or more the duration of the ventricular complex should be greater than 40 second we could not consider the heart action normal

Bazett has pointed out that the relation between the duration of systole and the heart rate can be expressed by the formula $QT = K\sqrt{cycle}$. If the heart cycle is determined from the measurement of several beats it can be written as R R. The Q T interval should also be measured in several beats and the formula can be written $K = \frac{Q\,T}{\sqrt{R\,R}}$. Bazett found the average value for K to be 0.37 for men and 0.40 for women.

Cheer and Li and Shipley and Hallaran have also investigated the value of K and have obtained somewhat different figures as follows

TABLE V

		MINIMUM	AVERAGE	MAXIMUM
Bazett	Male		370	
	Female		400	
Cheer and Li	Male	360	374	386
	Female	372	388	401
Shipley and Hallaran	Male	337	397	433
	Female	380	415	450

The differences in these measurements are probably due to differences in *methods* of measurement of the Q T duration as it is sometimes difficult to decide upon the point at which the T wave ends. This value K has been called the systolic index and has been found to vary under the influence of disease, particularly with dilatation of the heart, and under the influence of drugs.

Ashman and Hull have studied this constant and feel that the formula $Q\,T = 0.385 \times \log 10\,(R\,R + 0.07)$ is better than the above for males and suggest *that* the figure 0.375 be used for measurements in females.

It is impossible to choose between these formulas because of the probability that they all have certain defects and certain advantages. All agree that the Q T duration is shorter with increas

ing heart rate and that for the same heart rate the duration is longer in women than in men Whichever formula one uses and that of Bazett has had the most use at present, will be found a satisfactory basis for further investigation of accessory factors affecting this function of the heart

There is good evidence from experimental work that the duration of systole will be increased with increased diastolic filling of the ventricles even if the rate remains unchanged It is possible that the relation between heart rate and the duration of the ventricular complex may thus express the degree of cardiac dilatation at the various rates but this is as yet clinically unproved

The state of cardiac nutrition has been suggested as a possible cause of the variations in the duration of systole poor nutrition causing prolongation and vice versa This theory has some support from certain observations by Meakins but still awaits further proof

The U Wave

The U wave is associated with the recovery phase of heart muscle and comes at the time when the so called supernormal recovery phase might be expected (page 198) Ventricular premature beats are found to occur during the course of this wave especially when coupling is present When the heart rate is 100 per minute or more the U wave is obscured by the following P With slower rates it is found to follow the T wave and with very slow rates it takes its origin from the descending limb of T Its frequency has been variously reported by different authors depending upon the amplitude of the deflection which they have demanded before giving the wave a name Lewis and Gilder found it in 90 per cent of their series of normals, considering only the limb leads It is also frequent in records by precordial leads In limb leads its height varies from 0.1 to as much as 1.5 mm and its duration from 0.16 second to 0.24 second In precordial leads it may be as large as 2 mm being usually largest in Lead 4 R It is increased in size after exercise and at the end of a period of forced holding of the breath Its beginning has been observed by Papp to bear a fixed relation to the QRS group so that when the Q T duration is longer than 0.40 second it tends

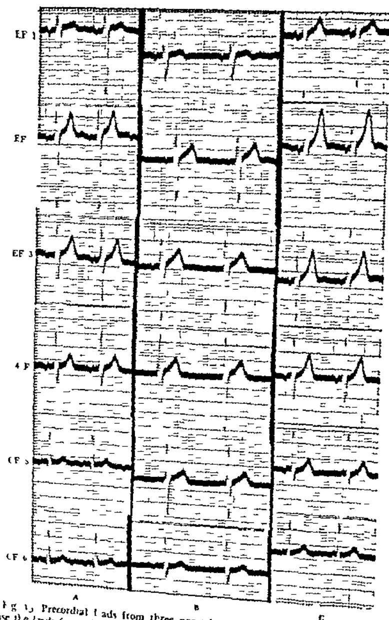


Fig. 13. Precordial leads from three normal individuals A, B and C. In each case the leads from above downward are from points E 1, which is in the sternal line to the right of the ensiform; F 2, which is in the sternal line to the left of the ensiform; E 3, which is half way between this and the next; 4, which is just to the left of the apex of the heart as determined by palpation, percussion and auscultation; C 5, which is at the level of position 4 but at the anterior axillary line; C 6, which is at the same level but at the midaxillary line. In each of these positions the electrode is paired with the left leg, which is indicated by the letter F. Note that there is a progressively increasing height of R from position 1 to 4 or 5, after which it decreases. As these were normal hearts, positions 5 and 6 were increasingly remote from the heart itself.

to fuse with the descending limb of T This may result in an apparent great prolongation of the T wave It has not been found inverted in records from normal hearts either in limb leads or precordial leads

NORMAL PRECORDIAL LEADS

The description of the normal variations of the waves of precordial leads is even more difficult than the description of the normal variations of the waves obtained by the standard leads because few normal hearts have been systematically studied and especially because different authors have not always used the *same precordial point or the same site* for the indifferent electrode when making their studies of normal persons The features of a curve obtained from the fifth interspace near the sternum differ radically from those of a curve obtained from just beyond the apex irrespective of where the indifferent electrode may be placed and there are known to be minor variations dependent upon whether the indifferent electrode is placed upon right arm left arm or leg The reasons for this are discussed in Chapter IX

Figure 15 is a series of records obtained by Leads EF 1 to CF 6 the technique for these leads has been described in Chapter I It is seen that R becomes progressively larger as we pass from right to left reaching a maximum which is usually found at Points 4 or 5 and after this diminishing S is usually maximum at Point 2 and becomes progressively smaller at points further toward the left It may often disappear at Points 4 or 5 A small Q may be found at Point 4 and often at Point 5 also T is usually largest at Point 2 or 3 and becomes progressively smaller as the electrode is moved toward the left

A series of records from 30 normal subjects obtained from precordial points 1 to 5 has been reported by Kossmann but since he used the central terminal described by Wilson as the indifferent point his measurements are not exact criteria for records obtained from the same points when the leg is used for the indifferent electrode His Leads are V_1 V V_3 V_4 and V according to the terminology suggested by the committee of the American Heart Association The values however would probably not differ by more than a few millimeters from those ob

tained when using the leg as the indifferent point Table VI contains measurements from his article and shows the maximum minimum mean and standard deviations of the various deflections he obtained It also shows the maximum minimum and mean values for the various waves obtained by Lead 4 F in 200 cases and by Lead 4 B in 21 cases by Shipley and Halloran and the values obtained using the right arm and left leg for the indifferent electrode and two precordial points in a group of 115 normals consisting of children and adults reported by Sorsky and Wood Table VII shows the values obtained by Barnes in records from 50 normal men and 50 normal women using Leads CF 1 to CF 6 Shanno has determined the values of the deflection of P, total QRS Q R S S T and T waves in 100 normal young women and has reported his results in such a way as to give an idea of the frequency of the different values Further studies giving the frequency of these and other measurements in normals are greatly needed *He found a Q wave only once in CF 4 but in 20 per cent of records by Lead CF 5* It never exceeded 2 mm in amplitude *The S T* junction was elevated in 17 per cent but never *more* than 2 mm In general his maximum and minimum values agree with those of Barnes for females as shown in Table VII

The P wave in precordial leads is usually better marked in the *region of the* sternum than toward the apex It may be directed upward or downward or may be *diphasic in* records from normal individuals *In leads* using the right arm for the indifferent *electrode P is usually larger* and is more likely *to be upright* than in leads using the leg

It will be noted from the tables that an R wave is almost always present although *it may be* very tiny and occasionally absent in records *from the right* of the sternum and occasionally less than 2 *mm* in records from *the left of the* sternum In leads from the apex with the *indifferent electrode* placed upon the back R may be as *small as 1 5* mm or 1 mm (Table VI) Barnes found the minimum value *of R to be less* than 2 mm in all six positions in records from *females but only* in the first three positions *in* males The knowledge *of* the small values obtained *in* females is of greatest importance because small values of R in precordial leads have been stressed as a sign of myocardial dis

TABLE VI

MEASUREMENTS OF WAVES IN PRECORDIAL LEADS OF NORMAL SUBJECTS

Lead		P			Q			R			S			S T			T		
		M n	Max	Mean	Min	Max	Mean	Min	Max	Mean	Min	Max	Mean	M n	Max	Direction of Deflection	Min	Max	Mean
IV B (30 S bject S tting)		0 2	1 0	0 68	—	—	—	* 1 0	* 28 0	* 13 0	* 1 5	* 24 0	* 10 0	—	—	—	2 0	14 0	6 9
IV F (00 Subjects Sitting)		0	1 4	0 45	0	3 0	—	* 5 0	* 23 0	* 11 1	* 1 0	* 25 0	* 9 4	−0 6	2 0	—	0 5	13 0	4 5
(30 Subject S p)	V1	—	—	—	0	0	0	1 0	9 6	4 16	3 4	24 0	11 05	−1 5	1 5	—	−4 0	5 6	1 23
	V	—	—	—	0	0	0	4 0	0 8	9 05	3 0	38 8	16 23	−1 5	1 5	—	2 4	11 0	6 27
	V	—	—	—	0	0 4	0 013	6 0	54 6	16 7	0	22 0	9 05	−1 5	1 5	—	3 6	12 0	6 26
	V	—	—	—	0	3 0	0 37	12 2	46 0	22 31	0	16 0	5 32	−1 5	1 5	—	2 4	11 0	5 66
	V	—	—	—	0	3 4	0.57	8 8	33 0	18 78	0	9 6	1 93	−1.5	1.5	—	2 0	9 6	4 59

Ape m§	0	25	14	05	30	15†	15	230	158	20	1 0	74	−10	20	+4 −47 i 63	30	140	69
Ape κ§	−10	5	+10 1 d 8		—	—	20	180	105	10	170	87	−10	12	+33 −5 i 75	05	150	40
L P 1 m§	05	0	08	10	15	‡	20	250	113	40	1 0	98	−10	20	+61 i 53	20	150	52
L Pect 1—1 g§	−20	10	+45 −1 d 15 o 24	—		—	10	150	64	40	200	118	10	−20	+53 i 41	−35	140	+85 −13 d 16

* Tw ty-o bi ts. ly
† Abse t 89 t r es
‡ O ly p sent tw
§ As tl t th sed the old w n g th d rect n I th d fl t on has been r sed in m k g th t bl Total ses 114
t l ca d sta mes ol d flect on
Ord ry um 1 n t t 1 re t on I d flect n of th w v The gn + or − d c t that th leff ct I d ect t pward or low a 1 fro n the
Th fi ure n it l cs rel t 1 re t on I d flect n of th w v The gn + or − d c t that th leff ct I d ect t pward or low a 1 fro n the b se l th numbe t are affected be ng d ated by the ume l f g e g +8 −8 di n t e t d pha c d fl tion is i tes soel tne th
n m ral fo l w ng in ach c se f d t k th n mbe of c se aff cted

TABLE VII

MEASUREMENTS OF WAVES IN PRECORDIAL LEADS OF NORMAL SUBJECTS (AFTER BARNES)

		P			Q			R			S			S T			T		
		M n	Max	Dir ction of Deflection	M n	Max	Mean	Min	Max	Me n	Min	Max	Mean	Min	Max	Mean	Min	Max	Mean
CF 1	Male	−1 7	5	+ 5 −36 di 9	0	0	0	0 2	9 8	3 3	6 6	35+	17	−0.5	1 7	+43 − 4 iso 3	0 2	6 7	+16 −29 di 5
	Female	−2 0	0 3	+ 1 −38 di 11	0	0	0	0 2	6 5	2 1	6 7	33 4	17 8	−1 0	1 3	+45 – so 3	0 3	3 9	+ 5 −36 di 9
CF 2	M le	−1 3	1 1	+13 −25 d 1	0	0	0	1 1	16 8	8 4	7 0	35+	25 4	0 2	3 5	1 9	3 1	15 5	7 1
	Female	−1 7	2 6	+ 6 −26 di 18	0	0	0	0 2	10 7	4 6	12 0	35+	24 5	0	2 4	1 3	1 7	10 1	4 3 di 1
CF 3	Male	−1 3	1 1	+13 −17 di 20	1	1 6	1 4	1 3	18 7	9 2	6 5	35+	21 1	0 5	3 4	1 8	2 8	14 9	7 5
	Female	−1 4	0 6	+ 7 −23 d 19	0	0	0	1 6	13 0	5 3	8 1	35+	19 7	0	2 5	1 2	1 2	9 3	4 8

CP 4	M l	−1 2	1 1	+17 / d 17	0 4	1 7	0 8	2 8	30 8	11 8	1 4	35+	14 5	−0 1	3 0	+19 / − 1	0 8	14 3	7 0
	F m l	−1 1	0 6	+ 7 / − 1 / d	0	0	0	1 1	39 0	7 5	2 2	2 3	14 5	0	2 6	1 0	1 3	9 3	5 5
CP 5	M l	−0 9	1 1	+10 / − 6 / d	0 1	1 0	0 7	4 7	35+	16 4	1 8	22 0	7 4	−1 1	2 0	+30 / − 2 / 8	0 7	12 3	5 4
	F m le	−0 9	0 6	+ 7 / −11 / d 1	0 1	1 0	0 5	1 8	24 9	12 0	8 6	18 9	5 8	−0 8	1 4	+33 / − 6 / 11	1 2	8 9	4
CP 6	M l	−0 8	1 3	+1 / −11 / d 0	0 1	2 4	1 0	3 2	26 0	13 4	0 1	7 6	2 5	−0 6	1 0	+21 / −13 / d 16	0 4	9 6	2 8 / di
	F m le	−0 4	1 8	+ 6 / − p / d 15	0 1	1 4	0 4	1 0	10 4	9 9	0 1	8 9	2 0	−0 7	0	+ 2 / −11 / di 17	0 6	6 1	2 8

M [illegible] obtained by D[illegible] on 50 normal ma[illegible] and 50 normal f[illegible] by l[illegible] from th[illegible] precordial points suggested by the Am[illegible] an H[illegible]
A [illegible] paired with the leg The pat[illegible] se ted
The n[illegible] al r[illegible] to mm [illegible] deflection [illegible] those [illegible] which [illegible] at the numb r [illegible] ses
Th[illegible] gn + or − wh[illegible] wed by [illegible] tes that the deflection [illegible] upw[illegible] or downwa[illegible] from th[illegible] The number [illegible]
ca es [illegible] cted be [illegible] ated by the n[illegible] +8 −8 d[illegible] and at[illegible] diphasic deflection [illegible] isoelectric the numeral follow[illegible] the
[illegible] mbe of ases [illegible] ffect d
Abs[illegible] t 13 t m s • Abs[illegible] t 4 t m Absent tw Ab[illegible] nt 14 times.

case particularly of infarction When a small R is normal however it will be accompanied by a large S a feature which does not appear in the table Without a large S a small R is less likely to be normal

The apex of the R wave of precordial leads is a point of great importance for it marks the time of arrival of the contraction stimulus beneath the precordial electrode (page 322) Its occasional absence in records from the right of the sternum is considered due to the fact that *in this position the electrode is not* over ventricular muscle but over the A V orifice The time of its occurrence may be measured from the beginning of the QRS group of the precordial lead and it will be found in normal hearts that it usually occurs earlier in records from near the sternum than in those from the apex or beyond The difference in time usually does not exceed *0 02 second* Sometimes the difference is very slight and occasionally there is no difference in the time of appearance of R in records from these different positions The time from the beginning of QRS to the peak of R does not exceed *0 01 second in normal records* even in Leads *I F and CF 5*

The duration of the QRS group of precordial leads is approximately the same as that of the duration of the widest QRS of the standard leads This matter however has not been carefully investigated Notching or slurring of QRS is commonly *found in* precordial records from normal hearts especially *in* leads from the region of the interventricular septum and cardiac apex where it *is* thought to result from the interference of the electric effects approaching the surface of the muscle beneath the electrode through the right and left ventricles simultaneously Curves from the right of this region do not show notching unless the electrode *is placed to the right of the sternum nor do* curves obtained to the left of the apex

The S T junction occurs at a point somewhat further from the base line than in the standard leads Shipley and Hallaran using Lead I F found an upward deflection *in over half of the records* with the maximum deflection of 2 mm They found a downward deflection very rarely (2 per cent) and the maximum deflection was 0 6 mm Sorsky and Wood and also Barnes usually found the

S T junction above the isoelectric level in leads from the apex sometimes as much as 2 mm It was occasionally below the iso electric reaching a maximum in this direction of 1 mm The contour of the S T segment is usually slightly convex toward the base line as it passes to the peak of T It usually begins almost immediately at the S T junction although occasionally after a short level interval In 4 records of Shipley and Hallaran s series the curve from the S T junction to the peak of T was diphasic (—+) at first slightly downward before turning upward toward the peak *

As in the standard leads the first limb of the T wave is usually concave upward the peak of T is further from its beginning than from its ending and the final limb of T is more steep than the first limb and may be almost straight although showing a gradual curve concave upward at its fusion with the base line The T wave is directed upward except in leads from the right sternal margin where it is occasionally isoelectric diphasic or downward Sorsky and Wood found occasional records with down wardly directed T waves in the left pectoral left leg lead which has the precordial electrode well to the left of the sternal margin Their series included many children and electrocardiograms of normal children have been frequently noted to show a down ward T in leads from the left of the sternum but not in leads *from* the apex Barnes did not find any downward T waves to the left of the sternum but found one record with diphasic T in CF 2 in a female

The Q T duration of *precordial* leads is not apparently differ ent from that of the standard leads although this has not as yet been studied carefully *Notching of the T wave* probably is not a normal phenomenon It is more commonly seen in records from diseased hearts Precordial leads usually show a well developed U wave following T It is apt to be more prominent than in the standard leads and in records from normal hearts is always di rected upward

* Their records as well as those of Sorsky and Wood were taken with reversed wiring so that their normal T wave was directed downward The above descrip tion however applies to the records they would have obtained had they used the wiring recommended by the American Heart Association so that the normal T wave is upward

It is believed for reasons which are elaborated in Chapter 1 that more than one precordial lead should be taken and that for ordinary diagnostic purposes precordial positions 2 3 4 and 5 should be used paired with the left leg

Precordial leads in children In children the QRS group of precordial leads tends to show a smaller R and larger S in leads

TABLE VIII

SUMMARY OF FEATURES OF NORMAL ELECTROCARDIOGRAM IN LIMB LEADS

	P	QRS	S T*	T
Height in largest lead	0 7 to 2 5 mm †	5 to 20 mm †	+1 3 to −1 mm	1 to 6 5 mm †
Direction Lead 1	Upward	Chiefly upward (R) Q or S *may be present* either singly or together but neither as large as R	Usually upward	Upward
Lead 2	Upward	Chiefly upward (R) Q or S may be present *either singly or together* but neither as large as R	Usually upward	Upward
Lead 3	Upward diphasic or downward	Q S or both may equal but not exceed R QRS group may be of vibra*tory type*	Upward or downward	Upward diphasic or downward
Form	Rounded may be notched	One two or three sharply pointed peaks or vibratory group *A peak may be notched in* a lead of relatively small excursion or near base line	Usually concave toward *peak of T*	Peaked
Duration	Not more than 0 10 sec	Not more than 0 10 sec	Variable usually short	Q T varies with heart rate 0 32 to 0 42 sec

P R interval varies with heart rate 0 12 to 0 20 second
* Measured in relation to P R level if this differs from zero
† In records where one lead is diphasic or isoelectric and the amplitude in the other lead about equal the normal values for P vary from 0 6 mm to 2 mm for QRS from 4 7 mm to 17 5 mm *and for T from 0 8 mm to 5 6 mm*

from the apical position than is usually found in records of adults The R however usually reaches a size of at least 7 mm In children less than 14 or 15 years leads from near the sternum often show an inverted T wave but Lead 4 F will always show an upward T wave Lead EF 3 or CF 3 will sometimes show an inverted T wave but less frequently than FF 2 or CF 2

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Luderitz B Uber Beziehungen zwischen der Breite von QRS und der Form

It is believed for reasons which are elaborated in Chapter 1 that more than one precordial lead should be taken and that for ordinary diagnostic purposes precordial positions 2 3 4 and 5 should be used paired with the left leg

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Form	Rounded may be notched	One two or three sharply pointed peaks or vibratory group A peak may be notched in a lead of relatively small excursion or near base line	Usually concave toward peak of T	Peaked
Duration	Not more than 0 10 sec	Not more than 0 10 sec	Variable usually short	Q T varies with heart rate 0 32 to 0 42 sec

[illegible] 0 12 to 0 20 second

othe l ads about mm to 17 5 mm

CHAPTER III

HYPERTROPHY OF THE CHAMBERS OF THE HEART

THE results of hypertrophy of one or another chamber of the heart afford the most frequently observed electrocardiographic abnormalities Hypertrophy of the muscle is not to be considered as an evidence of myocardial disease for it may arise from purely mechanical causes which demand an increased propulsion of blood by the chamber affected

As examples of this hypertrophy of the left ventricle may be caused by high blood pressure or disease of the aortic valve and right ventricular hypertrophy by congenital narrowing of the pulmonary artery or increased pressure in the pulmonary circuit due to mitral disease Besides the mechanical causes of hypertrophy there is what might be called a pathological cause If the muscle of a ventricle is diffusely affected by disease its power to propel blood will fail blood will accumulate within it the demand upon it will be thereby increased and a hypertrophy will result The electrocardiogram of such a heart will be affected by the hypertrophy but also by the disease so that it is usually possible to distinguish from the record whether a heart is hypertrophied by purely mechanical causes and has a practically sound muscle or whether disease of the muscle may be a cause of the enlargement

We shall consider in this chapter only the changes in the electrocardiogram which arise from hypertrophy of the muscle and not those due to its disease

AURICULAR HYPERTROPHY

The determination of auricular hypertrophy from the electrocardiogram rests upon rather meager correlations of clinical and pathological material Clinical and electrocardiographic correlations have indicated that such auricular hypertrophy as is associated with mitral stenosis usually results in a P wave in limb

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left auricle alone did not show an abnormal height of P Marked increase in the amplitude of P (more than 3 5 mm) was much more frequent when there was tricuspid as well as mitral valvular disease and the P waves of these cases were usually wider and more markedly notched

The author has observed clinically two cases believed to have uncomplicated tricuspid stenosis and both of these showed high sharply peaked P waves without abnormal duration With pulmonary stenosis the P wave shows an increased voltage and often *an increased* duration Winternitz has pointed out that hypertrophy of the right auricle is usually associated with a small though upright P_1 and a large amplitude *of* P_2 *and* P_3 *so that* P may reach a height of 3 or 4 mm Notching and increased duration are not he believes caused by right auricular hypertrophy *The above observations seem to point to the conclusion that* increased height of P probably is the result of hypertrophy *of the right auricle* that increased height duration and notching are probably the result of the hypertrophy of both *auricles It must* be remembered *that the height* of waves is subject to physiological *variations* and that with a seriously failing heart the same *heart might* not show as large an amplitude of P *as it would* were the muscle in a better *state of nourishment*

of

or more electric potential could be produced when the muscle is *increased in thickness* and that thus the height of P would be *increased It* is also understandable that it would take longer for the contraction wave to spread throughout the enlarged auricles *especially the large* left auricle so that the duration of P which represents *the time occupied* by this spread ing would be prolonged over the normal limits The notching can be explained on the *basis of the hypothesis* mentioned on page 56 for if the auricles are unequally enlarged their potentials will be more likely *to develop* to a maximum at *different* times causing a double peak or notching

In these records with *unusually* large P waves it is common *to* note that the P R level *is further* below the zero level than normal This deflection as has been *pointed out* is due to the auricu

leads with three features conspicuously frequent (1) The height of P is excessive being found 2 mm or more in the lead having the largest excursion in 75 per cent of any large group of records

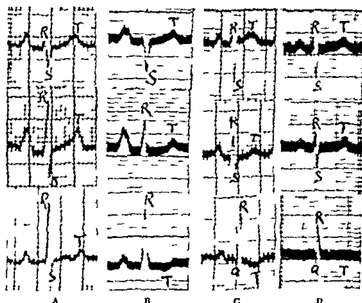


Fig 16 Records from four different hearts to show varying degrees of right axis deviation of QRS A indicates the least degree and D the greatest Note that S increases in size in relation to R as the degree of deviation increases In C and D S shows a progressive increase in its size

Records A and B each have a large and wide P wave indicative of auricular hypertrophy B has a plainly notched P wave in each of the three leads

from hearts with mitral stenosis and normal rhythm (Fig 16 A and B) The wave is sometimes found as large as 5 mm (2) The duration of P is excessive being over 0 10 second in 85 per cent of the records (Fig 16 A B and C) (3) Notching of P (Fig 16 B) occurs with unusual frequency being found in about 60 per cent of records from hearts with mitral stenosis and in only about 30 per cent of records from supposed normals

Master and Berliner have reported a small series of cases of uncomplicated mitral stenosis proved at autopsy The records showed a P wave of 2 mm or more in one half of the cases notching in almost all and widening in somewhat less than half The cases which had the large P waves (2 to 3 5 mm) were shown to have hypertrophy of the right auricle as well as hyper trophy of the left auricle those cases with hypertrophy of the

It is possible to determine the direction of this electrical axis from the relation of the size of the largest QRS excursion found in any lead to the size of the corresponding deflections in other leads This may be done roughly by means of Figure 21 With records like those of Figure 11 when QRS shows its chief deflection upward in all three leads the values may be represented as Lead 1+ Lead 2+ and Lead 3+ This places the electrical axis of these QRS groups within the sector lying between 30° and 90° within which sector we find the electrical axis of QRS in the great majority of normal hearts

When the direction of the axis of QRS points more toward the patient s right hand records like those of Figure 16 will be produced with the chief deflection of QRS downward in Lead 1 and upward in Lead 3 The sector (Lead 1— Lead 2+ Lead 3+) in Figure 21 will be found to represent the deflections of these records but other records will be found with deflections lying in the sector still further to the right (Lead 1— Lead 2— Lead 3+) When the electrical axis of QRS lies within either of these sectors it may be said to be deviated to the right or to *show right axis deviation*

When the direction of the axis of QRS points more toward the patient s left hand records like those of Figure 17 will be produced with the chief deflection of QRS upward in Leads 1 and 2 and downward in Lead 3 The sector (Lead 1+ Lead 2+ Lead 3—) of Figure 21 will be found to represent the deflections of these records Other records will show deflections which are represented by values found in the sector still further to the left (Lead 1+ Lead 2— Lead 3—) When the electrical axis of QRS lies within either of these sectors it may be said to be deviated to the left or to show *left axis deviation*

When following records of an individual case over a period of time it may be desirable to be more exact and determine the angle of the electrical axis by the method of Einthoven (Appendix 1) but for general clinical diagnosis a grouping according to the sectors of Figure 21 is quite adequate The progressive reciprocal variation of the values in the three leads which result from variations in the direction of the angle of any potential are shown in the figure but will be best appreciated if the first por

lar T wave and *since it is prolonged into the ventricular complex* as far as about the beginning of the rise of the T wave it is likely that it is responsible for the fact that certain of these records show this portion of the T wave definitely below the base line This can be observed in Leads 1 and 2 of Figure 16 A and B as well as in other illustrations throughout this book showing an unusual departure of the P R interval from the base line

Ventricular Hypertrophy

Hypertrophy of one or the other ventricle has a very definite effect upon the QRS group producing the changes which Lewis speaks of as a sign of right or left ventricular preponderance meaning that the right or left ventricle is *preponderantly* hypertrophied This use of the word preponderance has the fault of overstressing the importance of the weight of the ventricles When the term was introduced the ventricular weight ratio was considered to be a more important determining factor in the form taken by QRS in the three leads than has been shown by experience to be the case For this reason it seems better to express these variations of the QRS group by some term which is referable only to this group

It is pointed out in Chapter IX that the varying direction of the waves of QRS in each lead is due to the fact that there is a development of electrical potential within the heart which is continually varying both in direction and in magnitude throughout the duration of this group of waves At some time during the course of QRS there will be inscribed in one or another lead of most records a deflection which is larger than any other This is usually due to the heart potential of greatest magnitude * and this potential has been called the *electrical axis of QRS* Certain records may have two equally large excursions in adjacent leads that is Leads 1 and 2 or Leads 2 and 3 These usually represent the same heart potential and indicate the potential of the electrical axis of QRS

* An exception to this might occur occasionally when the greatest potential had a direction at right angles to one of the leads so that only 87 per cent of its value was recorded in the most favorable lead In this record some other heart potential which was not quite so large might actually give a larger excursion in the recorded electrocardiogram if its direction were parallel to a lead

deviation of QRS As the degree of axis deviation increases S_3 shows an excursion which is an increasingly large percentage of R_1 until it may equal R_1 or even exceed it as in records C and D

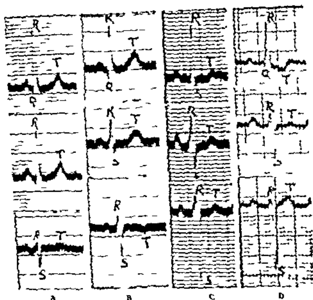


Fig 17 Records from four different hearts to show different degrees of left axis deviation of QRS A has the least marked deviation and D the most Note that S_3 increases in size in relation to R_3 as the degree of deviation increases until in D S_3 is larger than R_3 In C and D S_2 shows an increasingly large deflection In D QRS has a duration of 0 1 second

S_1 is often absent from these records This will be seen to be the reverse of the behavior of S_1 and S_3 in the presence of right axis deviation of QRS With increasing left axis deviation R_2 becomes relatively smaller and S_2 relatively larger (*Fig 17 C* and D) just as occurs with marked right axis deviation Such a Lead 2 might be part of a record showing either right or left axis deviation and without the other leads we could not say which it was for example compare Lead 2 of Figures 16 C and 17 C When S_3 gives the largest excursion of QRS in any lead and S_3 approaches the size of R_1 we have an extreme grade of left axis deviation (Fig 17 D)

Q is usually present in Lead 1 of records showing left axis deviation of QRS When it is prominent in Lead 3 the record should be suspected of indicating an abnormal spreading of the

tion of Einthoven's table on page 408 is reviewed In this table and in Figure 21 all values are made proportional to a maximum value of 10

Right axis deviation of QRS will be present when R_3 is larger than R_2 and there is a downward deflection in Lead 1 Occasionally there will not be an R wave in Lead 1 only a downward deflection called QS If R_2 and R_3 are equal it will indicate an electrical axis on the borderline of normal (Lead 1 = 0 Lead 2+, Lead 3+) It is better to consider the relative size of the deflections of QRS in Leads 2 and 3 than to consider the relative heights of R and S in Lead 1 because the larger deflections of R_2 and R_3 represent the predominant electrical forces and it is usual for these peaks to represent approximately the same instant in the QRS group whereas R_1 and S_1 may occur earlier or later and be related to lesser electrical potentials

Figure 16 shows four records A B C *and* D *with increasing* degrees of right axis deviation of QRS As the degree of axis deviation increases S_1 shows an excursion which is an increasingly *large percentage of R_3 until it may equal R_3 or even exceed* it as in records C and D of the figure As S_1 becomes relatively larger R_2 becomes a smaller fraction of the largest excursion of *QRS and S_2 tends to become larger A Q* wave is very rarely found in Lead 1 of records showing right axis deviation and when so found it usually indicates an abnormality of the spreading of the contraction *which has been produced by* cardiac disease Q is commonly found in Lead 3 and may reach a considerable size in this lead It may be present also in Lead 2

Further right axis deviation of *QRS leads* to an increase of S_1 and S_2 at the expense of R_1 and R_2 until eventually R_3 is found more than twice as large as R as in records C and D of the figure Such a marked degree of deviation to the right is uncommon

Left axis deviation of QRS will be present when R_1 is larger than R and Lead 3 shows S larger than R It is usual for the R wave in Lead *3* to be small and occasionally *it may not be* present so that there is only a downward deflection called QS Occasionally the downward deflection in Lead 3 will be found to precede R in which case *it will be called Q (Fig 16* B C D*)* In Figure 17 are four records with increasing degrees of left axis

system and in the position of the heart as it lies within the chest have been discovered to have an important influence upon this feature of QRS as has been pointed out in Chapter II so that a few records from normal hearts are found to show slight degrees of right or left axis deviation of QRS The effects of disease upon the branches of the bundle of His are such as to completely destroy the relationship between hypertrophy and the electrical axis of QRS

Effect of Position of Heart

It will be well here to discuss further the effect upon the electrocardiogram of the position of the heart in the chest We have pointed out that this can modify the record varying the relative size and direction of the waves in the three leads It acts in this way upon all the waves but most upon the QRS group and least upon P The influence upon the electrocardiogram of the changed position of the diaphragm during expiration can be well seen in Figure 18 where Lead 3 shows a gradual decrease in the size of R with coincident increase in the size of S It will be seen that as S_3 increases in size the relation of R_1 and R becomes more definitely that of left axis deviation

The amplitude of T_3 becomes smaller reaches zero and eventually a slightly downward T appears The increase observed in the voltage of the QRS group and of T with increasing left axis deviation during expiration is believed due to the uncovering of the heart by the lung margins allowing better contact of the heart with the anterior chest wall

These variations in the electrocardiogram are due to the movement of the diaphragm making the heart lie more transversely at full expiration and more vertically within the chest when the diaphragm sinks with inspiration The heart is relatively fixed at its base by the attachments of the arteries and veins and the apex is relatively movable so that the rise and fall of the diaphragm rotates the longitudinal diameter of the heart upon the base as a fixed point It will move as we view the patient from in front in a counterclockwise direction during expiration and in a clockwise direction during inspiration The heart will rotate upon its long axis at the same time twisting so that the anterior

contraction. If Q_3 is large (see page 137) in the presence of left axis deviation, the QRS group is considered to be abnormal, though cardiac disease is not necessarily implied.

When the electrical axis of QRS is deviated toward the right or toward the left of normal, such changes are usually due to hypertrophy of the right or of the left ventricle, though occasional normal hearts will give records showing this sign (page 41). It is necessary, however, that the ventricle should be hypertrophied in excess of any hypertrophy of the other ventricle which may coexist. It must be a preponderant hypertrophy, and this was the origin of Lewis' terms *right* or *left ventricular preponderance* as applied to this electrocardiographic feature.

Hypertrophy of the right ventricle only tends to produce records like those of Figure 16 when the left ventricle does not increase proportionately at the same time. The change is due to *a predominance of the electrical effect of one ventricle over that of the other*. For example, in Lewis' series a heart with ventricular weights of left = 158 gm. and right = 98 gm., the L/R ratio* being 1.61:1, which is a borderline right preponderance, showed an electrocardiogram with the QRS group as follows:

Lead 1	R = 1 mm.	S = 4 mm.
Lead 2	R = 5 mm.	S = 3 mm.
Lead 3	R = 6 mm.	S = 1 mm.

This is an example of slight right axis deviation of QRS similar to record A of Figure 16. Another heart with ventricular weights of left = 105 gm. and right = 128 gm., the L/R ratio being 0.82:1, which is a marked right preponderance, gave a record with the QRS group as follows:

Lead 1	R = 0.5 mm.	S = 8 mm.
Lead 2	R = 8.0 mm.	S = 5 mm.
Lead 3	R = 11.0 mm.	S = 3 mm.

This is an example of moderate or marked right axis deviation similar to records B or C of Figure 16.

The direction of the electrical axis of QRS will be affected by other things than hypertrophy of the right or left ventricle, and particularly if there is not much cardiac enlargement. Variations in the distribution of the branches of the auriculoventricular

* The normal L/R ratio varies between 1.5:1 and 2.2:1.

the anterior surface Both the movements of the long axis of the heart and the rotation upon its long axis take part in producing the changes which are seen with respiration The auricles move least so P is least changed The QRS group is most changed and T not so much as QRS

A fluoroscopic examination during respiration may sometimes reveal the change in the direction of the anatomical long axis of the heart to be as much as 30° The coincident rotation on the long axis can usually also be observed Obviously the electrocardiogram by the three leads must vary if the heart moves as much as this for if anything but a circle should rotate as much as 30° it would be sure to have a different appearance when viewed from a constant direction such as is represented by each of the leads (Chapter IX)

In few hearts however does the anatomical axis vary as much as 30° with respiration the usual rotation being about 15° Often the diaphragmatic excursion is slight and often the heart especially if of the vertical type will rest so lightly upon the diaphragm as to be scarcely affected by its motion Respiratory variation of a wave appears most plainly in whichever lead gives it the smallest relative value for in this lead the rotation of the heart can more easily change the wave from an upward to a downward deflection or vice versa This is why the vibratory type of QRS is especially likely to show definite variations with the respiratory movements

If the diaphragm is permanently high in the thorax a condition usually associated with a broad hypersthenic type of chest (Fig 19) or with an obese abdomen the heart will be permanently rotated so that its long axis will lie horizontally and the ventricles and septum will be rotated as with left ventricular hypertrophy The effect on the waves of the electrocardiogram will be to change their proportional value in the three leads so that P and T are likely to be relatively small in Lead 3 or T may even be inverted The QRS group is likely to resemble those of records C or H of Figure 11 or even record A of Figure 17 the latter indicating a slight left axis deviation of the QRS group If a heart which had a ventricular weight ratio with a balance

surface moves toward the right as the diaphragm rises with expiration, thus tending to produce a relationship of the ventricles and the septum to the chest wall which is similar to that pro

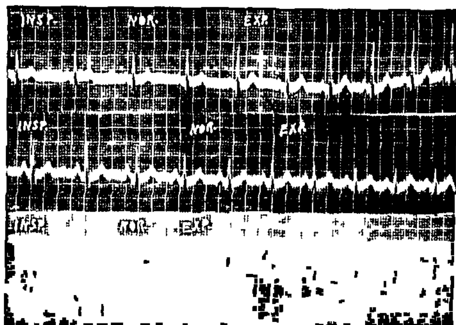


Fig 18 Electrocardiogram by three leads to show the changes in the waves resulting from forced inspiration and expiration At the beginning of each lead the chest was in the position of full inspiration During the lead the breath was exhaled *and at the end forced expiration was used The complexes underneath* the portions marked normal (*NOR*) are typical of those found in the records of this patient when breathing naturally It will be seen that at the left of the figure during inspiration the QRS group shows an electrical axis within the normal zone At the right during the expiratory portion of the figure the electrical axis of QRS is markedly toward the left The voltage of the QRS group increases during expiration

The T wave also changes during expiration becoming smaller in Lead 3 and larger in Lead 1 that is its electrical axis is also rotated somewhat toward the left The voltage of T changes but slightly but is larger during expiration

duced by left ventricular hypertrophy The anterior interventricular groove is displaced toward the right so that the left ventricle is more visible on the anterior surface of the heart With inspiration as the diaphragm moves downward the heart rotates so that the anterior surface moves on its longitudinal axis toward the left and produces a relationship of the ventricles and the *septum to the chest wall like that produced by hypertrophy* of the right ventricle The anterior interventricular groove is displaced toward the left so that the left ventricle becomes less visible on

the balance toward the right, yet still within normal limits, might give an electrocardiogram like Figure 16 A, showing slight right axis deviation of QRS. One with a ventricular weight ratio with

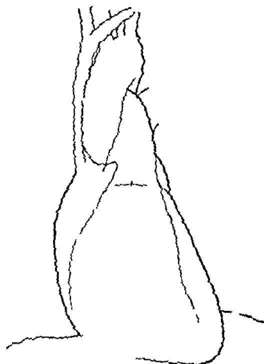


Fig. 2. Silhouette area of the heart of a relatively slender man. (After Bardeen, American Journal of Roentgenology and Radium Therapy, [illegible] 1927.)

the balance too far toward the left, just beyond the normal, might give a record of the type of D, F or E of Figure 11, not indicating the left *preponderance* which was present.

When either ventricle is *more than* slightly hypertrophied so that the relation of its *weight to the* other is decidedly abnormal, the influence of this *preponderance* combined with the changed relation of the two *ventricles* to the anterior plane of the chest will affect the *QRS* group strongly enough *to over*balance the *effect of an abnormal position* of the heart or an unusual distribution of the *bundle* branch tissue within the ventricles. Slight degrees of *mass preponderance* may only serve to counterbalance the effect *of an opposite* type of QRS group

slightly toward the right should be associated with a high diaphragm the slight *preponderance* of the right ventricle might be concealed by the effect of the position of the diaphragm and the



Fig 19 Silhouette area of the heart and great vessels of a stocky man Inside the silhouette area the ventral surface of the heart and the position of the valves have been schematically represented (After Bardeen *American Journal of Roentgenology and Radium Therapy* 9 827 1922)

QRS be normal Likewise a normally balanced heart with a high diaphragm would tend to show a QRS with slight left axis deviation and one with a ventricular weight ratio slightly toward the left yet within normal limits would be certain to show a definite left axis deviation of QRS

A permanently low diaphragm such as is usually associated with a long narrow chest has just the reverse effect on the waves of the record The heart hangs more vertically as shown in Figure 20 and the effect on the record is that P and T are likely to be relatively small in Lead 1 and QRS to have characteristics like those of records A and B of Figure 11 or even like Figure 16 A T will never be turned downward in Lead 1 by this position of the heart as it may be in Lead 3 by a transverse position In the long narrow thorax a heart with a ventricular weight ratio with

Attempts to Express the Electrical Axis of QRS

Various attempts have been made to ascribe an exact numerical rating which would indicate the degree and the direction of deviation of the electrical axis Einthoven determined the direction within the body of the potential which produced the largest excursion of the QRS group His method of determining the angle of any potential within the body from the values derived from the three leads is detailed in the Appendix He did not however indicate how the QRS group of certain unusual records should be treated neither those in which the values of correspondingly named peaks in the three leads did not fulfill his formula nor those with an equally large but oppositely directed QRS deflection in Leads 1 and 3 nor those with the largest deflection occurring twice in the same lead but in opposite directions as when R = S In the last two categories if the synchronous portions of the QRS group are determined for the other leads there will be found to be two electrical axes in quite different directions

In records with the highest peaks of the QRS groups occurring at the same instant in the three leads so that the height of Lead 1 + that of Lead 3 equals that of Lead 2 it is easy to apply Einthoven s table or Dieuaide s graph to determine the direction of the current within the heart (Appendix) In records with the high peaks not representing the same time instant of QRS so that they are out of phase the above formula will not be found correct for the peaks in the three leads do not represent the same potential within the heart even though they may have the same name It is necessary by inspection of the QRS group in the other two leads to determine the part of QRS in each lead which coincides in time with the peak of the largest deflection It may be helpful to measure under low magnification the distance from the beginning or the ending of QRS to this peak and to measure the deflection in other leads at the same time after the beginning of QRS or preceding its ending When measurements are obtained which approximately fulfill Einthoven s formula 1 + 3 = 2 it is satisfactory to derive mathematically the smallest value from the two

which may have resulted from an abnormal position of the heart or from an unusual distribution of bundle branch tissue. For example a vertical heart whose normal electrocardiogram is like that of Figure 11 A would need more mass preponderance of the left ventricle to produce a left axis deviation of QRS than would a transverse heart whose normal record was like that of Figure 11 F. With more markedly preponderant hypertrophy of either ventricle the features of position and bundle branch distribution become of diminishing importance. As a result of this an enlarged heart which gives a record of either right or left axis deviation is much more likely to have this because of a muscle preponderance than is a heart with the same degree of electrocardiographic variation but without enlargement.

The effect of lateral displacement of the heart. When the patient turns on the left or on the right side there is to be seen a change in the electrical axis of QRS and of T. Turning the patient on the left side marked changes appear giving *rise to a rotation of the electrical axis of QRS toward the right* so that R becomes smaller and S larger in Lead 1 and Q and R become larger in Lead 3. The electrical axis of T is less affected than that of QRS. When the patient turns on the right side similar though less marked changes appear. This change with position may be due to the heart falling to one side in the chest as the patient turns and thus changing the anatomical axis of the heart. Along with this change in the anatomical axis there is a certain amount of rotation of the heart on the longitudinal axis which will also contribute to the changes observed *in the waves*.

A similar tendency toward right axis deviation appears when the heart is displaced to the left as a result of pulmonary tuberculosis either by fibrosis or by pneumothorax. When the heart is transposed as is found with congenital dextrocardia the electrocardiogram shows an inversion of all of its waves in Lead 1 while the waves of Leads 2 and 3 resemble those of an ordinary record of right axis deviation of QRS. In this way acquired and congenital dextrocardia can be differentiated if other signs are not more evident.

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larger ones and discard the actual measurement of the smallest excursion

At times one cannot determine the coincident time phases in the three leads with sufficient exactness without a record of two leads taken simultaneously When this is necessary and not available the following procedure will suffice to obtain an approximation sufficiently accurate for most purposes

When the two largest waves of QRS occur in Leads 1 and 2 or in Leads 2 and 3 it is usual to find these two peaks representing the same instant of the QRS group— in phase —and their values should be used to derive the values of the other lead For example Figure 16 A gives $R_3 = 13$ mm $R_2 = 11$ mm Hence

$$QRS_1 = R_2 - R_3 (11 - 13) = -2 \text{ mm}$$

Figure 17 A gives $R_1 = 15$ mm $R_2 = 12$ mm Hence

$$QRS_3 = R_2 - R_1 (12 - 15) = -3 \text{ mm}$$

When the two largest waves are found in Leads 1 and 3 it will *not be possible to predict which peaks will fall at the same time* instant We must try to decide this by inspection of the record with magnification measuring from the beginning of QRS of each lead to the respective peaks It is not usual in such records for R_1 and S_3 to occur at the same time so that the larger of the two *should be used in combination with the similarly named* peak of Lead 2 When R_1 and S_3 are of equal value (or S_1 and R_3) it is best to consider that Lead 2 = 0 Such records as has been said have two electrical axes of equal voltage which differ considerably from each other

Lewis suggested a method of obtaining an index of ventricular preponderance by the formula $(R_1 - R_3) + (S_3 - S_1)$ the height in millimeters of the waves in the leads being indicated by R_1 S_1 R_3 and S_3 He did not mention what he considered to be the normal limits for the result of this formula expecting probably to determine this by a sufficient number of autopsies with weighing of the separated ventricles White and Bock suggested a very similar formula $(U_1 + D_3) - (D_1 + U_3)$ in which U is the height in millimeters of the largest upward deflection of QRS and D is the height of the largest downward deflection in the leads indicated by the figures The result of this formula will

differ from that of Lewis formula only when Q is larger than S in either Lead 1 or Lead 3

Lewis formula is more sound theoretically than White's for the Q and the S waves of Leads 1 and 3 are apparently governed by different ventricles and not by the same ventricle as they are considered to be in White's formula In an attempt to determine what the normal limits might be for Lewis formula the author reviewed 30 cases published in part by Lewis and in part by Cotton The ventricular weights and electrocardiographic records of these patients were compared and for hearts without preponderant hypertrophy of either ventricle the index figure usually lay between +20 and —10 A figure greater than +20 then should indicate left ventricular *preponderance* and one less than —10 right ventricular *preponderance* White states that +30 or more indicates left axis deviation with a border zone between +20 and +30 and that —15 indicates right axis deviation with a border zone of —10 to —15

These formulas have two obvious faults (1) It is difficult to apply a proper numerical correction for displacement of the long axis of the heart The angle of Einthoven lends itself more readily to such a correction (2) Their most important defect is that the index is dependent upon the actual amplitude of the waves as recorded in the three leads to such an extent that when this becomes very small it is not possible to obtain large values of the index Figure 10 A for instance with measurements of Lead 1 = +2 Lead 2 = +1 Lead 3 = —1 would have the electrical axis of QRS according to Einthoven's method definitely to the left of normal that is $\alpha = 0$ This record gives an index of +2 [(2 + 2) — (1 + 1)] which is not beyond the normal range and yet if the measurements were multiplied by 15 [(30 + 30) — (15 + 15)] the index would be +30 which is definitely to the left of normal

Figure 17 B which indicates definite left axis deviation of QRS according to Einthoven's method that is alpha = +0° (Lead 1 = 11 Lead 2 = 6 Lead 3 = —5) gives an index figure of 14 [(11 + 6) — (1 + 2)] which is within the limits of normal

Schlomka adopting a formula similar to that of White and Bock attempted to correct it for the error resulting from the

variations in the amplitude of the excursions in different records His formula is

$$\frac{(O_1 - U_1) - (O_3 - U_3)}{(O + U)\ \text{max}}$$

In this formula O and U are the values of the largest upward and largest downward deflection of the QRS group in Lead 1 for the first parenthesis and Lead 3 for the second parenthesis The value of (O + U) max is obtained from either Lead 1 or Lead 3 depending upon which of these leads gives the largest figure for the sum of the largest upward and largest downward deflection *He suggested that positive values indicated a predominance of the left heart* and negative values a predominance of the right heart but had not determined at what point the heart index should be considered to indicate an abnormal predominance

Carter Richter and Green suggested subtracting the chief upward and downward deflections in Lead 1 to obtain a value for Lead 1 and subtracting the chief upward and downward deflections of Lead 3 to obtain a value for Lead 3 The values so obtained were then used to determine by a geometric graph devised from the formula of Einthoven the direction of the electrical axis which would have produced such values for Leads 1 and 3 This in effect determines the algebraic sum of the upward and downward deflections of these leads and bases *the electrical axis upon these derived figures* There is doubt as to what such an algebraic sum represents and greater doubt as to what is represented by the electrical axis derived from two such figures

Recently the Criteria Committee of the New York Heart Association has suggested a method of determining the deviation of the electrical axis of QRS Upward peaks are to be regarded as positive and downward peaks as negative The algebraic sum of the QRS deflections of each of the three leads is to be determined There is considered to be *no deviation* of the electrical axis of QRS if this value is positive or zero in Lead 1 positive in Lead 2 and positive or zero in Lead 3 *Left deviation* of the electrical axis is said to be indicated when the

algebraic sum of the QRS deflection is positive in Lead 1 and in Lead 2 negative in Lead 3 If it is negative in Leads 2 and 3 the degree of left axis deviation is to be considered marked Right deviation of the electrical axis is considered to be present when the algebraic sum of the QRS deflections is negative in Lead 1 with a positive value in Leads 2 and 3 If Leads 1 and 2 both give a negative value the degree of the righ deviation is marked if negative in all three leads the degree of *right axis* deviation is more marked This method is similar to that of Carter Richter and Green in that it considers a mathematical mean value for each of the leads It does not deal with the electrical axis of any particular recorded potential It does not in fact derive an axis but assumes that the QRS group as a whole is expressed by the + or − value of the *algebraic summation of the* positive and negative deflections of each lead It is doubtful *if this is always* the case but the idea is based upon Wilson s suggestions as to the importance of the area of the QRS *group* (Chapter IX)

It is indeed unfortunate that so many *methods have been suggested to* express the relation *of the* QRS group to ventricular *hypertrophy* for none of them is capable of producing *figures* which can be closely correlated with definite *degrees of hyper*trophy The *figures* express something about the QRS group but this cannot be *transferred* with exactness either to hypertrophy or [illegible] relative preponderance *of the right or left* ventricular muscle masses *The* correlation with ventricular preponderance is *better when the* heart is large When it is of medium or *normal s ze the* correlation is very poor as has been shown particularly by *the* work of *Hermann* and Wilson On this account it has *seemed* to the author that for clinical purposes it will suffice to *view the QRS* group pictorially as it were and to determine the direction *of the axis of* the large[illegible] deflection of QRS by noting *the direction of the* [illegible]
in each l[illegible]
of Figur[illegible]
mately [illegible] *it is* better as he
[illegible] to compute the smallest from the two larg[illegible] by substituting

them in the above formula unless the smallest value should be in *Lead 2* In such cases, by using the vertical time lines carefully, measuring from the beginning of the QRS in each lead

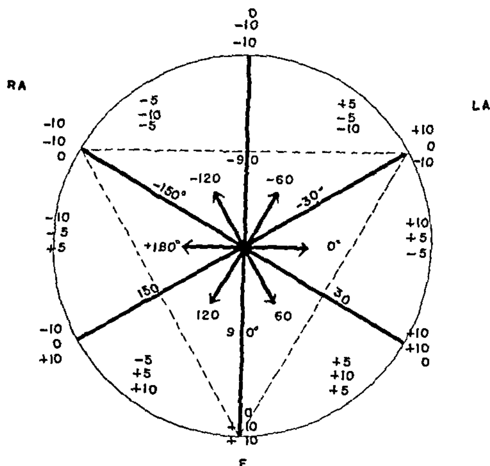


Fig 1 A scheme to represent graphically the direction of the deflections in the three leads resulting from any potential within the heart with a direction in this plane The circle is divided into six sectors when the potential under consideration has a direction parallel to any of the radii within a sector the direction of the deflections in the three leads will be as indicated by the (+) or (−) signs When the potential is parallel to the side of a sector one of the leads will show no deflection (= 0) and the other two will have equal deflections The direction at the center of each sector e g 60 in the + + + sector will produce two leads with equal values the remaining lead having a value exactly double The numerals indicate the proportional distribution of values in the three leads at the angles indicated if the maximum deflection is 10

we can often arrive at a very good approximation of coincident time points in the three leads

When the values are all upward (+) the leads may be represented by + + + and the angle of such a potential lies within

the sector (+30° to +90°) as may be seen by referring to Figure 21. This may be called the sector of normal balance for the QRS group. If Lead 1 or Lead 3 is small in relation to the others then the angle of the potential lies near the lower or the upper border of this sector. When the border is reached the lead will give a zero (0) value and this would constitute a borderline right or left deviation of the axis of QRS. We must now consider the long axis of the heart noting whether it is unusually vertical or transverse. If so we must take this into account in considering what significance in regard to ventricular hypertrophy is to be attached to the angle of the electrical axis of QRS. We can correct for the unusual position of the heart by rotating the angle of the axis slightly in the direction of the movements of the hands of a clock if the heart lies too horizontally and counter clockwise if the heart is too vertical.

A moderate right or left axis deviation of QRS will be shown by the values in the leads being − + + or + + − respectively with the negative value always less than the value of Lead 2, for example the measurement of Figures 16 A and 17 A.

16 A Lead 1 = −2.5 Lead 2 = +10.5 Lead 3 = +13
17 A Lead 1 = +17 Lead 2 = +11.5 Lead 3 = −5.5

If the value in Lead 2 becomes equal to or less than the value in Lead 1 in records with right axis deviation as in Figure 16 B

16 B Lead 1 = −6 Lead 2 = +6 Lead 3 = +12

then we may consider the axis deviation of marked degree. If Lead 2 approaches zero or becomes a minus value as in Figure 16 C or Figure 16 D

16 D Lead 1 = −6 Lead 2 = −0.5 Lead 3 = +5.5

the degree of deviation should be considered as very marked.

In records showing left axis deviation of QRS the condition is analogous, Figure 17 B indicating a moderate degree and Figure 17 C indicating a marked degree of left axis deviation.

17 B Lead 1 = +10.5 Lead 2 = +5 Lead 3 = −5.5
17 C Lead 1 = +12 Lead 2 = −4 Lead 3 = −16

In practice the measurements may be fitted pictorially into the scheme of Figure 21.

Voltage of QRS. The height of the largest recorded wave of

QRS in whatever lead this may be varies in general with the degree of ventricular hypertrophy tending to be large if a marked right or left axis deviation is shown It is also on the average larger in generally enlarged hearts which show a normal direction of the axis than in hearts of normal size Both of these statements are made with the qualification that the physiological condition of the muscle should be equally good or equally poor in the hearts compared and that there is no myocardial abnormality which would distort the QRS group or extracardiac condition to produce unusual short circuiting of the heart s current A heart with the muscle in a poor physiological condition will tend to have small waves no matter what the degree of hypertrophy so that this factor will often reduce to normal or less the size of the excursions of the QRS of a much hypertrophied heart The effect of an abnormal path of the contraction wave due to myocardial disease is not predictable (Chapter iv)

Increased duration of the QRS group In many records with marked left axis deviation the duration of QRS is increased to more than 0 10 second The prolongation of QRS in many such records is due to the increased thickness of the ventricular muscle through which the contraction wave must pass in going from the *endocardial to the pericardial surface We know from experi*ments upon dogs and from readings on the human heart that the last part of the ventricular muscle to become involved in the contraction is at the basal part of the left ventricle Since QRS lasts until the whole of the ventricular muscle has entered into contraction it is readily understandable that thickening of the left ventricular muscle may cause a prolongation of QRS The rate at which the contraction passes through the ventricular wall of the dog is 5 mm per 0 01 second If it were the same rate in man and it is likely that it is slower then an added centimeter of thickness of the ventricular wall would prolong QRS for 0 02 second

We should not then consider a duration of 0 11 second abnormal for the QRS of records showing marked left axis deviation and if the heart is very large or the axis deviation very marked as in Figure 17 D even 0 12 second may be due to the hypertrophied ventricular wall and not to disease of the con

ducting tissues Excluding from consideration cases of bundle branch block which we know have a wide QRS because of a delayed conduction to one ventricle a general relationship has been demonstrated between the weight of the heart and the duration of QRS

Hermann and Wilson however have produced evidence to show that even when the left ventricular wall is much thickened the duration of QRS need not be prolonged Price and Pardee also have found that even very large hearts may have a normal duration of QRS From all these observations it seems that though a prolongation of QRS to 0 12 second may at times be due to an increased thickness of the left ventricular wall yet in other hearts it may be due to a slightly delayed conduction in the left bundle branch (incomplete bundle branch block see page 128) such as might result from slight depression of the function of this tissue Such a disturbance of the function of the bundle branch would be expected to cause a marked left axis deviation of QRS with a prolonged QRS duration and an inverted T_1 as in Figure 17 D Without inversion of T_1 incomplete bundle branch block probably should not be diagnosed

When the duration of QRS is greater than 0 10 second in association with right axis deviation we may conclude that in all but exceptional cases there is delayed conduction in the right bundle branch Thickening of the wall of the right ventricle does not reach a sufficient degree to cause a prolongation of the QRS group except in certain cases of congenital abnormality (Figure 39 C) In doubtful cases precordial leads may give information regarding the time of arrival of the contraction over different parts of the precordium (page 129) which will help to decide whether or not bundle branch block is present

The T wave The T wave is not changed by ventricular hypertrophy unless this is extreme and possibly not even then Many records with marked right axis deviation of QRS (Fig 16 D) or marked left axis deviation (Fig 17 C) show a T wave which is normal in all three leads In a large series of records with right axis deviation of QRS about 25 per cent showed a downwardly directed T and T_3 In a series with left axis deviation T_1 was inverted with or without T_2 with about the same frequency

Barnes and Whitten have found a similar relationship and have *suggested that these changes in the T wave are due to the* presence of ventricular strain They defined this as a physiologic myocardial disturbance due to the fact that the muscle is asked to perform work in excess of its metabolic capacity They believe that there is an increased hydrogen ion concentration and a shortened duration of the contraction in the ventricle affected The resulting electrical imbalance affects the T wave and the lead or leads affected are determined by the affected ventricle

Rykert and Hepburn studied 20 cases with marked left axis deviation and inversion of T_1 in 4 of these careful microscopic study failed to reveal any myocardial degeneration or fibrosis so that hypertrophy was the sole abnormal feature of the muscle Rykert and Hepburn pointed out three other features which are frequently found in electrocardiograms associated with marked left ventricular hypertrophy (1) The S T junction is definitely below the isoelectric line in Lead 1 and usually above the iso electric line *in Lead 3 In Lead 1 the S T segment passes to the* peak of T with an upward convexity which sometimes rises suffi ciently above the level of the S T junction to suggest a coronary T wave In Lead 3 the S T segment is concave upward and is not unusual in appearance (2) The voltage of QRS tends to be large (3) The voltage of T tends to be large Recently Barnes has mentioned these features of the S T segment as additional signs of left ventricular strain

In the case of records with right axis deviation the T wave sometimes shows a slight elevation of the S T junction in Lead 1 but without any other unusual feature In Leads 2 and 3 T is often *inverted (25 per cent)* and the *S T junction in Lead 3* is often somewhat below the isoelectric level the remainder of the S T segment being in no way remarkable and showing the usual convexity toward the base line commonly observed with an inverted T This is the electrocardiographic picture which Barnes and Whitten have attributed to right ventricular strain (Fig 39 A)

The one clinical condition which seems to afford a good ex ample *of acute right ventricular strain* is embolism of a branch of the pulmonary artery After this occurrence the electrocardio

gram does not tend to develop right axis deviation although it may develop a larger S_1 than was present previously The actual size of the S wave however may not be more than 3 or 4 mm A Q wave often develops in Lead 3 and may reach such a size that it is more than 25 per cent of the largest R deflection The S T segment usually is found to begin below the isoelectric line in Lead 1 In Lead 2 it may show an upward convexity as in the coronary T wave while T_3 is inverted and the S T segment although convex toward the base line may or may not show the coronary diphasic (+ −) appearance These changes it will be noted are in so many ways different from those attributed to chronic right ventricular strain that it is difficult to admit that they are the results of a similar process

The direction of the electrical axis of QRS however often is associated with a definite direction of T in the different leads though the direction of the axis is not apparently the sole determining factor Nor does inversion of T seem to depend upon the degree of deviation of the axis for a marked axis deviation may coexist with an upward T wave in all three leads Nor does it depend upon the degree of ventricular hypertrophy for a similar reason It seems as though the direction of the axis of QRS is a controlling factor so that when the contraction is affected by some other influence left axis deviation leads to an inversion of T_1 and perhaps also T while with right axis deviation T and T_3 become inverted Whether this influence is ventricular strain or a relative coronary insufficiency the muscle having outgrown the capacity of its arteries to supply its needs or a dilatation of the affected ventricle with resulting elongation of the bundle branch and slight delay in the spreading of the contraction to this ventricle is a matter still to be decided

Precordial Leads with Hypertrophy of Right or Left Ventricle

The variations of the precordial electrocardiogram associated with right and left ventricular hypertrophy have been studied but the variations due to right and left axis deviation have not as yet been distinguished from those due to hypertrophy It is likely that the variations of the QRS group at least are analogous

in the two conditions if not also the variations of T With right ventricular enlargement the QRS group tends to show a deeper S wave with all positions of the electrode and a smaller R wave than usual in records from the region of the apex and beyond *Whereas in normal hearts the peak of the R wave tends to fall* later in leads from the apex than in leads from the left sternal border as has been stated on page 72 records with right hypertrophy may show the R peak coincident in these two positions and occasionally even later at the right sternal border than at the apex *The T wave tends to be inverted most frequently in leads* from the right side of the sternum less frequently from the left of the sternum and occasionally in leads from the region of the apex

With left ventricular enlargement the QRS group tends to show an *especially* large R wave in leads from the apex and one which is smaller than usual and at times missing in leads from the left sternal margin The time difference between the peak of R in the sternal leads and in the lead from the apex is usually greater than normal in these records and increasingly so with the degree of hypertrophy The T wave is especially likely to be inverted in leads from the apex or beyond less likely in the parasternal lead and rarely in the left sternal lead

It is not certain whether the T wave changes of precordial leads from hearts with right or left ventricular enlargement are related to the variations of QRS or are due to some additional feature such as ventricular strain or deficient coronary flow or myocardial disease The situation is similar to that of the T wave changes in limb leads as discussed on page 99 It has been observed however that with left ventricular enlargement the T wave of the lead from the apex is only found inverted when there is a very large R wave in this lead

Wood and Seltzer have determined that in patients with left axis deviation due to upward displacement of the diaphragm it was usual to find inversion of T in precordial leads from the right of the sternum and not uncommon to find it in leads from the left of the sternum They found it rarely in leads from the apex They pointed out that the finding of an inverted T wave in leads from the right of the sternum may be of aid in dis

tinguishing between left axis deviation due to upward displacement of the cardiac apex by a high diaphragm and that due to significant enlargement of the left ventricle If T is upright in this lead no inference can be drawn but if it is inverted enlargement of the left ventricle is unlikely

Clinical Applications

Auricular hypertrophy will be suggested by the changes in P that have been described It may be associated with venous congestion in either the pulmonary or the systemic veins The former is far more common and the accepted electrocardiographic signs of auricular hypertrophy probably depend mostly upon a hypertrophied left auricle Mitral stenosis causes the most marked auricular hypertrophy but mitral regurgitation has a similar though less marked effect The effect of tricuspid stenosis in causing a P wave of large voltage has been mentioned Disease of the aortic valve will increase the work of the left auricle when the left ventricle fails to empty itself properly and the same is true of high arterial tension thus these conditions lead only secondarily to auricular hypertrophy

The finding of right or left axis deviation of QRS is not always an indication of the presence of ventricular hypertrophy the axis deviation may be due as has been said to minor congenital variations in the distribution of the bundle branch tissue and is somewhat influenced by the position of the heart in the chest the vertical type of heart tending to produce a right axis and the horizontal type tending to produce a left axis of QRS Deviation of the electrical axis of QRS may be associated with hypertrophy of both ventricles the one whose sign is most impressed upon the electrocardiogram being hypertrophied to a greater degree No it is uncommon to find a much hypertrophied heart whose electrocardiogram has a QRS group without either right or left axis deviation of QRS This ind[illegible]
the two ven[illegible]
has hype[illegible]
of their [illegible] [illegible]ering the significance of right or left axis deviation of QRS we must take into account the actual size of the heart and its position within the chest the

larger the heart, the greater importance is to be attached to a deviation of the electrical axis as an indication of hypertrophy of one or the other ventricle With smaller hearts the position or the peculiarities in the distribution of the contraction wave may be predominantly important Having observed the presence of absence of abnormal deviation of the electrical axis we must now attempt to appraise the size of each ventricle separately and to consider what might have been the cause of the hypertrophy which may be diagnosed

Hypertrophy of the right ventricle may be caused by disease of the pulmonary arteries with narrowing Chronic emphysema and chronic pulmonary tuberculosis often lead to hypertrophy of the right ventricle, the narrowed pulmonary capillary bed probably being the cause Certain congenital abnormalities of the heart cause great hypertrophy of the right ventricle Pulmonary stenosis patent interventricular septum and patent ductus arteriosus obviously would do this but a patent foramen ovale or a prominent ventricular band would not Congenital cardiac defects may also be accompanied by abnormalities of the branches of the auriculoventricular bundle so that at times the hypertrophy will be accompanied by an abnormal path of the contraction wave This will distort and vitiate the electrocardiographic sign of ventricular hypertrophy

Mitral stenosis leads to an hypertrophy of the right ventricle through a damming back of blood in the pulmonary capillaries thus increasing its work Mitral regurgitation does this also though secondarily and only after overtaxing the left ventricle Hence the earliest records with this valve lesion may show a left axis deviation of QRS later ones giving a normal QRS and later still as stenosis develops a right axis deviation

Hypertrophy of the left ventricle is caused by disease of the aortic valve either regurgitation or stenosis by arterial hypertension by coronary arteriosclerosis by mitral regurgitation and by diffuse myocardial degeneration Long continued strenuous physical exertion occasionally may lead to left ventricular hypertrophy though more often to an hypertrophy of both ventricles so that QRS is not changed Arterial hypertension and aortic stenosis cause the most marked left ventricular hypertrophy and

as they affect the right ventricle only secondarily through failure of the left they lead to marked grades of left axis deviation of QRS

Though the determination of a *preponderant* hypertrophy of either ventricle is not the most important information derived from the electrical curves yet it cannot be obtained in any other way and often throws an important light upon a doubtful diagnosis The presence or absence of hypertrophy of the right ventricle is often a factor in deciding whether the rumbling diastolic murmurs heard at the apex when aortic regurgitation is also present are due to a coincident mitral stenosis or to the aortic regurgitation by the mechanism described by Flint The presence of a right axis deviation is a proof that mitral stenosis is present though it is not uncommon to find a QRS with the normal relation of the waves in the three leads due to a balanced hypertrophy from a slight degree of stenosis combined with the aortic lesion

Two valvular lesions deserve special mention A pure uncomplicated mitral regurgitation will usually give an electrocardiogram showing a slight or moderate degree of left axis deviation or a normal ventricular relation irrespective of the amount [illegible]

[illegible] normal direction of the axis Most of these have little or no enlargement of the heart and are often without the increased pulse pressure so characteristic of this valve lesion Yet the murmur may be quite typical and often fairly loud Most of these patients have a vertical position of the heart which could mask a slight left side hypertrophy as previously explained In certain cases however it is impossible to explain the lack of abnormal axis deviation

The clinician is sure to wonder *how exact a measurement of relative ventricular size this electrocardiographic sign has been found to be* It is not particularly exact but it is liable to be more so the more definite are the evidences of cardiac enlargement Those hearts with a definite pathological *preponderance of* either ventricle usually give a definite axis deviation of the QRS group The discrepancies between the records and the ventricular

larger the heart the greater importance is to be attached to a deviation of the electrical axis as an indication of hypertrophy of one or the other ventricle With smaller hearts the position or the peculiarities in the distribution of the contraction wave may be predominantly important Having observed the presence *of absence of abnormal deviation of the electrical axis we must* now attempt to appraise the size of each ventricle separately and to consider what might have been the cause of the hypertrophy which may be diagnosed

Hypertrophy of the right ventricle may be caused by disease of the pulmonary arteries with narrowing Chronic emphysema and chronic pulmonary tuberculosis often lead to hypertrophy *of the right ventricle the narrowed pulmonary capillary bed* probably being the cause Certain congenital abnormalities of the heart cause great hypertrophy of the right ventricle Pulmonary stenosis patent interventricular septum and patent ductus arteriosus obviously would do this but a patent foramen ovale or a prominent ventricular band would not Congenital cardiac defects may also be accompanied by abnormalities of the branches of the auriculoventricular bundle so that at *times the hypertrophy will be accompanied by an abnormal* path of the contraction wave This will distort and vitiate the electrocardiographic sign of ventricular hypertrophy

Mitral stenosis leads to an hypertrophy of the right ventricle through a damming back of blood in the pulmonary capillaries thus increasing its work Mitral regurgitation does this also though secondarily and only after overtaxing the left ventricle Hence the earliest records with this valve lesion may show a left axis deviation of QRS later ones giving a normal QRS and later still as stenosis develops, a right axis deviation

Hypertrophy of the left ventricle is caused by disease of the aortic valve either regurgitation or stenosis by arterial hypertension by coronary arteriosclerosis by mitral regurgitation and by diffuse myocardial degeneration Long continued strenuous physical exertion occasionally may lead to left ventricular hypertrophy though more often to an hypertrophy of both ventricles so that QRS is not changed Arterial hypertension and aortic stenosis cause the most marked left ventricular hypertrophy and

as they affect the right ventricle only secondarily through failure of the left, they lead to marked grades of left axis deviation of QRS.

Though the determination of a *preponderant* hypertrophy of either ventricle is not the most important information derived from the electrical curves, yet it cannot be obtained in any other way and often throws an important light upon a doubtful diagnosis. The presence or absence of hypertrophy of the right ventricle is often a factor in deciding whether the rumbling diastolic murmurs heard at the apex when aortic regurgitation is also present are due to a coincident mitral stenosis or to the aortic regurgitation by the mechanism described by Flint. The presence of a right axis deviation is a proof that mitral stenosis is present, though it is not uncommon to find a QRS with the normal relation of the waves in the three leads, due to a balanced hypertrophy from a slight degree of stenosis combined with the aortic lesion.

Two valvular lesions deserve special mention. A pure uncomplicated mitral regurgitation will usually give an electrocardiogram showing a slight or moderate degree of left axis deviation or a normal ventricular relation, irrespective of the amount of hypertrophy of the heart. Uncomplicated aortic *regurgitation* is usually associated with left axis deviation, but there are many that show a normal direction of the axis. Most of these have little or no enlargement of the heart and are often without the increased pulse pressure so characteristic of this valve lesion. Yet the murmur may be quite typical and often fairly loud. Most of these patients have a vertical position of the heart which could mask a slight left-side hypertrophy as previously explained. In certain cases, however, it is impossible to explain the lack of abnormal axis deviation.

The clinician is sure to wonder how *exact a measurement of relative ventricular size this electrocardiographic sign has been found to be.* It is not particularly exact. For it is liable to be more so the more definite are the evidences of cardiac enlargement. Those hearts with a definite pathological *preponderance of* either ventricle usually give a definite axis deviation of the QRS group. The discrepancies between the records and the ventricular

weight ratios are usually of such a character that they might well have been due to a modification of the QRS group by a vertical or a transverse position of the heart within the chest At times an extreme variation of the electrical axis may be found without cardiac enlargement This usually is due to pathological changes in the muscle

SUMMARY

The electrocardiographic diagnosis of auricular hypertrophy may indicate a distinction between hypertrophy of the right and of the left auricle Hypertrophy of the right auricle is especially associated with a large voltage of P while hypertrophy of the left auricle is especially associated with increased duration and prominent notching of P

In the diagnosis of ventricular hypertrophy we are able to determine with fair accuracy whether the right or the left ventricle is chiefly affected The greater the cardiac enlargement, the greater our accuracy in this respect In making this diagnosis we must consider both the direction of the electrical axis of QRS and the direction of the anatomical axis of the heart as determined by a vertical or a transverse position of the heart within the chest

Hypertrophy of the right ventricle may be diagnosed with an enlarged heart if right axis deviation of QRS is found Without cardiac enlargement it should not be diagnosed if the heart is of the vertical type with a low diaphragm With a normal or transverse position of the heart and right axis deviation hypertrophy of the right ventricle is probable

Hypertrophy of the left ventricle may be diagnosed with an enlarged heart if left axis deviation of the QRS is found Without cardiac enlargement it should not be diagnosed if the heart is of the transverse type with high diaphragm With a normal or vertical position of the heart and left axis deviation hypertrophy of the left ventricle is probable

Hypertrophy of both ventricles may be diagnosed with an enlarged heart if there is neither right nor left axis deviation of QRS

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CHAPTER IV

CHANGES DUE TO MYOCARDIAL DISEASE AND FUNCTIONAL ABNORMALITIES

THE most important feature of electrocardiographic diagnosis depends upon the fact that myocardial disease can change the electrical manifestation of the heart beat and thereby produce abnormal waves in the electrocardiogram. Such electrocardiographic abnormalities will be present whether the heart is regular or irregular, fast or slow. They depend upon the abnormality of the muscle of the chambers concerned and therefore are found each time the chambers contract.

Beats arising from abnormal foci, such as premature beats and tachycardias, also produce abnormal waves. Such waves are readily distinguished by the presence of the arrhythmia from those due to disease of the muscle.

Functional variations in the muscle are also capable of causing changes in the electrical waves. The electrical production may be changed if the muscle is abnormal because of fatigue or of a deficient blood supply, or because of the action of drugs or of toxins. These are temporary variations, lasting only as long as the causative agent is active. If the cause is operative for a long period of time, structural changes may supervene. A single record may not suffice to tell whether an abnormality is due to abnormal function or to disease, but later records or a review of the clinical features of the case should enable us to decide.

ABNORMALITY OF THE P WAVE

Auricular premature beats or auricular tachycardia often result from auricular myocardial disease but may also be either toxic or neurogenic in origin. Auricular fibrillation or auricular flutter (Chapter VII) are usually due to structural disease of the auricles but also may be due to toxic or even to neurogenic

causes. Except for the implications from the finding of these arrhythmias little could be learned until recently about the auricular muscle from the changes in the form of the P wave.

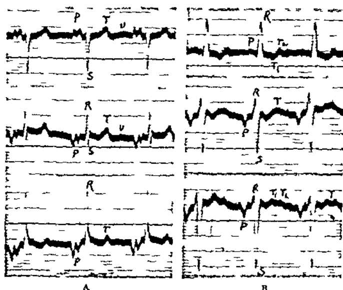


Fig. 22. A Abnormal P waves notched and with increased duration. The ventricular complexes show right axis deviation of QRS.
B Abnormal P waves inverted in Leads 2 and 3. The ventricular complexes show marked left axis deviation of QRS, a curious double-topped T wave in Lead 3, a diphasic T wave in Leads 1 and 2. The waves marked T 2 in Leads 1 and 3 and T in Lead 2 are in fact the U wave.

Very few clinical pathological correlations have been reported to justify the diagnosis of special pathological changes as the cause of P wave abnormality. The P wave sometimes presents a quite abnormal appearance, however, aside from the changes described as resulting from hypertrophy.

Abnormal P waves are seen in Figure 22 A and B, and such abnormal waves often persist. We believe this abnormality is due to a focal disease in the auricular muscle because marked notching of P or inversion in Lead 1 or in Leads 2 and 3 are the type of changes which would be expected to result from the abnormal contraction due to disease of the auricular muscle. Lambert has recently indicated a specific deformity of P as resulting from occlusion of auricular branches of the coronary arteries, namely, a double topped P wave or a diphasic (+ —) P or an inverted P. These features are best marked in Leads 2 and 3, as in Figure 22

A and B He emphasized also that in the acute stage there might be an inverted dome shaped deformity of the P R interval (auricular T) Master has emphasized the frequency of an increased height of P during the acute stage of ventricular infarction but the mechanism of its production is a matter of speculation at present since it does not seem to be confined to any particular site of the lesion It seems more likely to be due to some physiological change such as auricular dilatation than to pathological changes in the auricular muscle Unusually large P waves are also frequent in records from patients with toxic goiter

The only experimental work which has come to my attention bearing upon this is a single record published by Lewis to illustrate another matter in which marked notching and large size of P were present after one auricular appendix had been crushed Notching of P is the sort of change which might be expected to follow an injury of this sort Disease might in a similar way lead to an interruption of the normal path of the contraction wave or to an uneven spreading of the contraction to the right and left auricles and this would cause a notched P wave

Unless it leads to arrhythmia auricular myocarditis has little effect upon the functional ability of the heart for the force of the heart beat is in the ventricular contraction Myocardial disease is but rarely localized in the auricles but nevertheless an ability to diagnose auricular myocarditis may occasionally give a better insight into the pathological processes of certain patients in whom ventricular disease is either absent or is insufficient to change the ventricular curves

When the largest excursion of P recorded in the limb leads is less than 1 mm we have an indication of a poor state of nutrition a poor functional condition of the muscle This may be called low voltage of P Only 7 cases (10 per cent) in the combined normal series of 78 cases of Lewis and the author gave a measurement of less than 1 mm for the largest P wave and only 1 of these less than 0 7 mm An abnormally small P wave might also result from a diffuse disease of the auricular muscle in contrast to a focal process which would be more likely to cause a notched P wave Small P waves are seen after prolonged infec

tious diseases and in hearts which are diagnosed as having narrowed coronary arteries In both instances the factor of poorly nourished muscle may evidently be present The P wave has often been seen to increase in size during convalescence from infectious diseases and after recovery from cardiac failure and as both are occasions when the condition of the muscle is improving these observations are cited in further support of the hypothesis

Since a hypertrophied auricle will give a P wave of greater than normal size when its muscle is in good condition it will be rare for the P wave from such a heart to sink below the size which has been considered a minimum for auricles which are not hypertrophied

Abnormal Ventricular Waves

Each of the waves of the ventricular complex is subject to variation in height width and form as a result of abnormality of the contraction of the ventricular muscle The resulting ventricular complexes therefore may differ widely from one case to another and it is rare for the complexes from different patients to have more than the most general resemblance to each other The QRS group may be large or small increased in width or notched and along with these changes the T wave may be large or small or may be inverted in one or more leads Certain features however tend to occur together in special combinations so that we find certain types of abnormal ventricular complexes

Bundle Branch Block The most frequent combination of abnormalities of the ventricular complex appears to be ascribable to a special sort of pathological lesion A discussion of the mode of its production may well serve as an introduction to the discussion of the other changes in the ventricular complex resulting from disease

If in a dog pressure is made over the branch of the auriculoventricular bundle which carries the stimulus from the main bundle to the right ventricle the passage of this stimulus will be blocked The left ventricle then is the only one to be affected by the impulse from the A V node The left ventricle is the first to contract and the right ventricle then becomes involved by the

spreading of the contraction from the left ventricle through the interventricular septum With this change in the character of the spreading of the ventricular contraction the form of the ventricular complex immediately changes to one showing certain features seen in the records of Figures 23 and 24 which are from human hearts considered to have disease affecting one of the bundle branches and interfering with its function

The typical characteristics of these electrocardiograms as they are obtained from patients are as follows (1) the ventricular complex has an abnormally wide QRS group the duration being at least 0 13 second and usually 0 16 second (2) The QRS group usually though not always shows slurring or notching in more than one lead or there may be a notching of one or more of the peaks (3) The largest wave of QRS is usually oppositely directed in Leads I and 3 either upward in Lead 1 and downward in Lead 3 or vice versa such curves are said to be of the discordant type Certain records may have the largest wave of QRS in the same direction in Leads 1 and 3 either both upward or both downward these curves are spoken of as concordant types (4) If the P wave is present it is followed by the ventricular complex after a P R interval which is usually normal though occasionally varying degrees of heart block may be present If auricular fibrillation is present these bundle branch block complexes will occur with the irregularity characteristic of this condition as in Figure 24 Certain other special characteristics of these curves will be described under the captions of the various types of right and left bundle branch block complexes

The increased duration of QRS indicates that the contraction has taken an abnormally long time to involve the whole of the ventricular mass This is because it must now spread from one ventricle to the other through the ventricular muscle of the septum instead of being distributed to both ventricles almost simultaneously by the right and left branches of the bundle of His and their ramifications When the contraction has spread through the septum from one ventricle to the other it is distributed throughout the second ventricle by means of the branches of the auriculoventricular conducting tissue within that ventricle That is it passes from the muscle of the septum to the ramifications of

the conducting tissue and thence through this tissue to the remainder of the muscle of this ventricle The contraction passes through the ventricular muscle at a much slower speed than along the auriculoventricular conduction system In the dog's heart the figures are 500 mm per second for ventricular muscle versus 5000 mm per second for the conducting tissues During the normal human ventricular contraction the distribution of the impulse to the entire inner surface of both ventricles is probably completed in 0 02 or 0 03 second The remainder of the 0 08 or 0 10 second needed for the complete spreading of the contraction (shown by the duration of the QRS group) is occupied in its passage through the thickness of the ventricular wall It is evident from this that the spreading of the contraction from one ventricle to the other through the septum would greatly prolong QRS

We are able to determine from the direction of the chief deflections of QRS in the three leads which of the ventricles is the *first to enter into contraction From this we deduce that the* bundle branch to the opposite ventricle is affected

For many years the localization of the lesion in hearts showing bundle branch block was incorrectly stated because too close an analogy was drawn between the human electrocardiogram and the records obtained from dogs after cutting or crushing the branches of the bundle This erroneous conclusion was further established by a report of the pathological studies of two human hearts which gave electrocardiograms of this type Eppinger and Stoerck reported this study in a very incomplete form but concluded that they had found a lesion of the right bundle branch in two patients with records like those of Figure 24 A On the other hand Oppenheimer and the author in 1920 reported finding in the heart which gave the record of Figure 24 B a destruction of the right branch of the bundle and in the heart giving Figure 28 B a lesion of the left bundle branch with the right branch quite intact

Other authors particularly Mahaim and Yater also have investigated the microscopic pathology of the bundle branches of hearts which have given records of bundle branch block It is

common in these hearts to find evidence of abnormality in both branches of the bundle so that the interpretation of the functional result is difficult This difficulty is increased by the occasional finding of bundle branch block without evidence of gross interruption of the anatomical structure of either bundle branch It is evident that a bundle branch need not be entirely destroyed in order to have its function so depressed as to produce characteristic electrocardiographic alterations It is probable that when both branches are affected the more seriously affected branch determines the form of the electrocardiogram Even though the anatomical evidence for localization of the lesion is still somewhat confusing it has become increasingly evident with more numerous and more useful studies that the original localization of the site of the lesion was incorrectly stated

Additional evidence regarding the localization of the lesion producing bundle branch block has been obtained from the human electrocardiogram as a result of the study of the curves of ventricular premature beats obtained by direct electrical stimulation of the human ventricles (Chapter vi) Four separate groups of investigators are in practical agreement in attributing curves with the QRS group directed chiefly upward in Lead I to a premature beat starting in the right ventricle and curves with QRS directed chiefly downward in Lead I to a premature beat starting in the left ventricle Only occasional exceptions are noted (Chapter vi) It is probable that bundle branch block curves are more nearly uniform in their mode of development than are ventricular premature contractions because there is only one bundle branch in each ventricle so that the contraction stimulus has only this one mode of entry into the ventricle when the other branch is blocked Premature beats on the other hand may start in a multitude of different situations so that the stimulus may enter the ventricle at one of many sites The localization of premature beats by the direction of QRS in Lead I supports the newer concept of the localization of the lesion responsible for bundle branch block Certain observations make it probable that both premature beats and bundle branch block curves may be modified by the presence of a preponderent ventricular hyper

trophy so that the general rules of localization may not apply in the specific instances

Kountz has added further evidence in favor of the newer localization by cutting the bundle branches of revived human hearts The newer more carefully controlled animal experiments also have contributed results which indicate that the older ideas of the localization of the lesion of bundle branch block were incorrect Wilson and his associates have studied direct leads from the ventricle of the dog and also the human precordial leads, and have applied the results to an analysis of the curves of bundle branch block It is largely through this work that the present localization has come to be accepted so it will be discussed here in some detail

As is pointed out in Chapter IX (page 322) the time of arrival of the contraction stimulus beneath the precordial electrode is indicated by the peak of the R wave of precordial leads This observation made it possible to confirm the localization of the lesion in curves showing bundle branch block by determining over which ventricle this peak was developed earliest and therefore which first entered into contraction Simultaneous records of Lead I and of the precordial leads were obtained and the time *between the occurrence of the peak of R_1 and the peak of R in* the precordial lead was measured It was found that in the cases giving records now considered to indicate right bundle branch block the peak of the R wave occurred much later in curves from the sternum and the left sternal border than in curves from the apex and beyond (Fig 29) This delay of the R wave over the right ventricle they attributed to block of the right bundle branch Likewise they found that in cases giving records of the type now considered to indicate left bundle branch block the peak of the R wave was much earlier in the precordial leads obtained from the sternum and to the left of the sternum than in leads from the apex and beyond (Fig 30) This indicates an earlier arrival of the stimulus in the right ventricle and consequently *left bundle branch block Such great differences in the time of* the peak of the R wave at different precordial points are not observed in records with a QRS group of normal duration

Left bundle branch block curves may be divided into two gen

eral types depending upon whether the chief deflection of QRS is opposite in Leads 1 and 3 (discordant) or in the same direction (concordant) in these leads Besides the abnormal duration of the

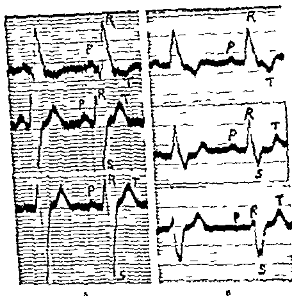


Fig 23 A and B indicate that the left branch of the auriculoventricular bundle is not functioning—left bundle branch block Note the abnormal width of the QRS group the notching of QRS and the T wave directed opposite to the chief QRS deflection in Leads 1 and 3 The normal P R interval of the records shows that the function of the main auriculoventricular bundle is normal

QRS group which must be at least 0 13 second the characteristic features of these two types of curves due to left bundle branch block are as follows

1 Discordant type (Figs 23 A and B 27 A) *Lead 1* the chief deflection is R though there may be a small Q The T wave is usually downward occasionally upward *Lead 3* the chief deflection is S though there may be a smaller R T is upward

2 Concordant type (Figs 23 B 30 C) *Lead 1* is the same as described for 1 a A

though ther

downward

In either type the S T segment may be displaced above or below zero the direction of this displacement being the same as that of the peak of T

The curves of *right bundle branch block* have been divided into four types by Bayley, and Wilson has subsequently described a fifth type. Besides the abnormal duration of QRS the

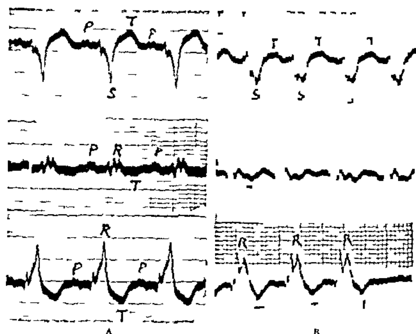


Fig. 24. Abnormal ventricular complexes indicating right bundle branch block. The abnormal width and notching of the QRS group and the T wave opposite to the chief QRS deflection in Leads 1 and 3 are the important features. Record A has normal auriculoventricular conduction time. Record B shows auricular fibrillation. The microscopic examination of the heart which gave this electrocardiogram showed a destructive lesion of the right branch of the bundle.

characteristic features of these types of right bundle branch block are as follows:

Type 1 (Figs. 24 A and B, 28 A, 29 C). Lead 1, the chief deflection is a broad S with a smaller R. T is upward. Lead 3, the chief deflection is R which is broad and notched. T is downward.

Type 2 (Fig. 29 B). There is a broad S but R is larger than S. T is upward. Lead 3, the chief deflection is R which is broad and notched. T is downward.

Type 3 (*Figs. 25 A and 29 A*). *Lead 1, there is a large* R and a broad S. T is upward. Lead 3, there is a slender downward spike of considerable size sometimes preceded by a small upward deflection and always followed by a broad summit smaller in amplitude than the downward spike. T may be downward, isoelectric or slightly upward.

Type 4 (Fig 26 A) Lead 1 the chief deflection is a large R followed by a broad smaller S T may be upward or downward Lead 3 shows a small R followed by a broad deep inverted spike



Fig 26 Records showing bundle branch block A Right bundle branch block type 3 B Left bundle branch block of the concordant type

although occasionally the last part of this deep S wave may be isoelectric and merge with the S T segment T may be upward or downward

Type 5 (Fig 26 B) Lead 1 R and S are both present but both have small amplitude T may be upward or isoelectric Lead 3 a broad deep S T is upward

The S T segment is often displaced above or below zero in the direction taken by the peak of T although the displacement is not usually as great as is observed in records of left bundle branch block

The Nomenclature and *Criteria for Diagnosis of Diseases of the Heart* written by a committee of the New York Tuberculosis and Health Association has somewhat condensed this classification of right bundle branch block as follows

Type A corresponds to Type 1 as described above

Type B corresponds to Types 2 and 3 as described above

Type C corresponds to Types 1 and 5 as described above

As yet no special clinical features have been associated with the individual types of left or right bundle branch block The

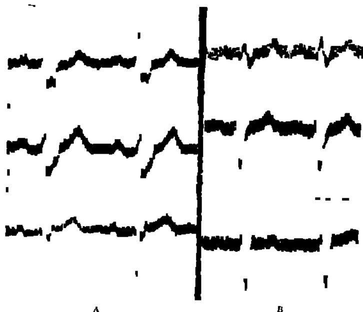


Fig 26 Records showing right bundle branch block A Ventricular complexes of the type 1 variety B Ventricular complexes of type 5

five types of right bundle branch block curves described by Bayley and Wilson can be fairly well distinguished in any large series of records showing these peculiar features The author recently found the following distribution in a group of 83 such records from the New York Hospital Type 1 31 Type 2 11 Type 3 33 Type 4 6 Type 5 2 The same New York Hospital series contained 74 instances of left bundle branch block so that the two bundle branches seem about equally susceptible to disease It is possible that the influences of a different position of the heart in the chest and of different varieties and degrees of ventricular hypertrophy may be responsible for the different types of curves of right bundle branch block and perhaps also for the different types of left bundle branch block

Figure 27 A and B and Figure 28 A are examples of another variant of these curves characterized by the small size of the excursions and the marked notching of QRS This type of complex was ascribed by Oppenheimer and Rothschild to arboriza

tion block this term intending to indicate a pathological process which involved the ramifications of the auriculoventricular system rather than the main right and left branches of the bundle

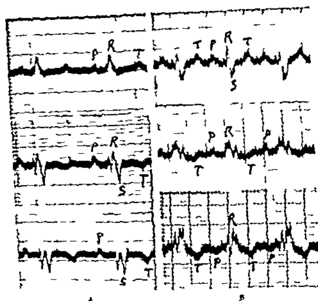


Fig 27 Record showing the features of bundle branch block combined with unusually small excursions of QRS

A A later record of the patient of Figure B showing changes in the complexes of the earlier record due to a change in the pathological lesion

The lesions giving rise to this type of curve were supposed to affect the terminal arborizations of the bundle branchings Such curves were frequently found with a degenerative process due to coronary arteriosclerosis and involving the subendocardial muscle fibers in the lower and anterior portions of the interventricular septum and the adjacent portions of the left ventricular wall It seems likely from subsequent clinical reports that such curves are not always obtained from hearts showing these pathological changes nor does this type of pathological change always give rise to this type of ventricular curve It is an occasional not a specific association but it is definitely an abnormal type of record and due to myocardial disease The abnormal features of these curves the notches and abnormal duration of QRS and the abnormal direction of T indicate an abnormal path of the

contraction through the ventricular muscle and the presence of bundle branch block plus some other factor to account for the low voltage of the waves. The smallness of these deflections may

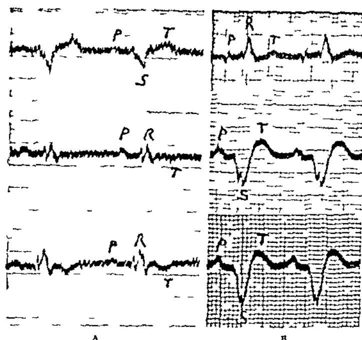


Fig. 28 A. A later record of the patient of Figure 24 A showing little change in the complexes except diminution in size.

B. Left bundle branch block. P-R equals 0.20 second. From a patient whose heart showed a sclerotic involvement of the left bundle branch, the posterior half of this branch being replaced by connective tissue continuous with an adherent mural thrombus.

be attributed to a poor condition of the ventricular muscle (pages 19 and 143). Figure 27 A is a later record of the patient from whom Figure 25 B was obtained and shows a change in form suggesting a further development of the lesion in the myocardium. Figure 28 A is a later record from the patient of Figure 24 A. The original form of the complex is in the main retained, the chief difference being in the size of the waves.

Notching of QRS is an evident feature of these last records and also of Figure 24 A, which has larger waves. The marked notching of these complexes (more than the single notch commonly found at the peak of R or S) as in Figures 23 B, 24 B and 25 B may be due to secondary irregularities in the spreading of the contraction wave resulting from the areas of myocardial degeneration so common throughout these hearts (Chapter V).

The *precordial leads* of patients with bundle branch block are also characteristic They show the increased duration of the QRS group and those of right and left bundle branch block are characteristically different With *right bundle branch block* (Fig 29) the curves obtained from near the sternum show the peak of the R wave much delayed occurring from 0 08 second to 0 10 second after the beginning of the QRS group The peak of R is frequently notched or there may be two upward peaks with a downward deflection between them forming an *M type complex* so that neither peak should properly be called the R wave In these records Wilson has shown by taking simultaneous records of Lead I and the precordial leads that the second peak represents the preintrinsic deflection indicated by R in the other precordial leads Leads from the left side of the heart in the region of the apex and beyond show a sharp R wave usually large though sometimes small whose peak occurs soon after the beginning of the QRS group sometimes as early as 0 02 second or 0 03 second The remainder of this QRS is composed of a broad slurred or notched S wave which is usually small though sometimes large

The precordial leads of patients with *left bundle branch block* (Fig 30) show in the leads from near the sternum a very small R wave or occasionally none at all The size of the S deflection obtained near the sternum is apt to be very large so that often t' [illegible] duced in order to r [illegible] The T wave also [illegible] usually large in these leads At or usually beyond the region of the apex the QRS group may show the upward and downward deflections of an M type of complex beyond the apex it shows an R wave which does not develop its peak until from 0 08 to 0 12 second after the beginning of the QRS group in that lead and often shows a slurring or notching on its ascending limb This R wave often is not well developed until as far lateral as the midaxillary line (Lead CF 6)

The time from the beginning of the QRS group to the peak of the R wave gives an indication of the time of appearance of the contraction stimulus at the various precordial points This seems to be as satisfactory a measurement as is the comparison of this peak with a fixed point in the record of another lead such as

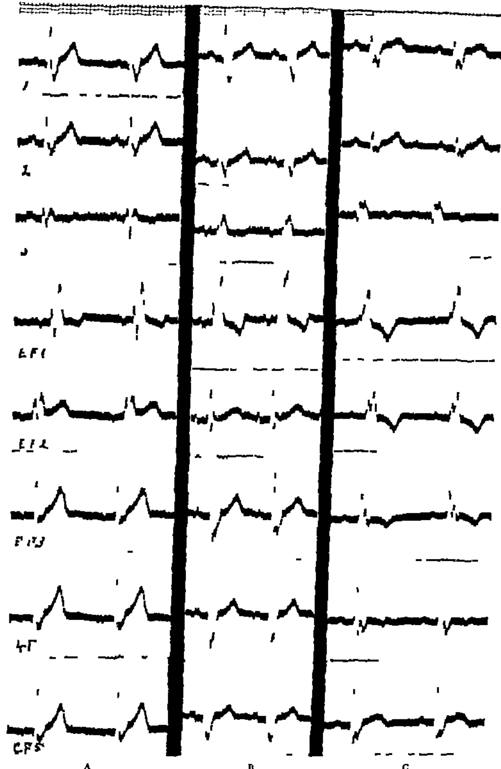


Fig. [illegible]9 Records indicating right bundle branch block. The three upper rows are the limb leads and below these precordial Leads EF 1, EF 2, EF 3, 4 F and CF 5.

A Bayley's type 3, B type 2, C type 1.

It will be noticed that the peak of the R wave appears late in Leads EF 1 and EF 2, occurring 0.08 second after the beginning of QRS except in record B which shows the delayed R only in Lead EF 1. Leads from points farther to the left show an early apex of R 0.02 to 0.04 second after the beginning of QRS.

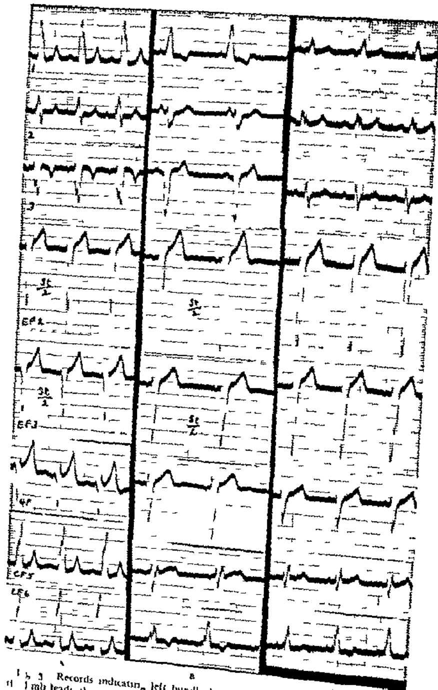


Fig. 3. Records indicating left bundle branch block. The first three rows are the limb leads, the rows below these being Leads EF 2, EF 3, 4 F, CF 5 and CF 6. St/2 indicates one-half standardization in the lead so marked, that is, 5 mm = 1 millivolt.

Records A and B are of the discordant type, record C of the concordant type. Note the early appearance of the peak of R in Leads EF 2 and EF 3 and in Lead 4 F of records B and C. Leads CF 5 and CF 6 and Lead 4 F of record A show a later appearance of the peak of R, this occurring from 0.08 to 0.10 second after the beginning of QRS.

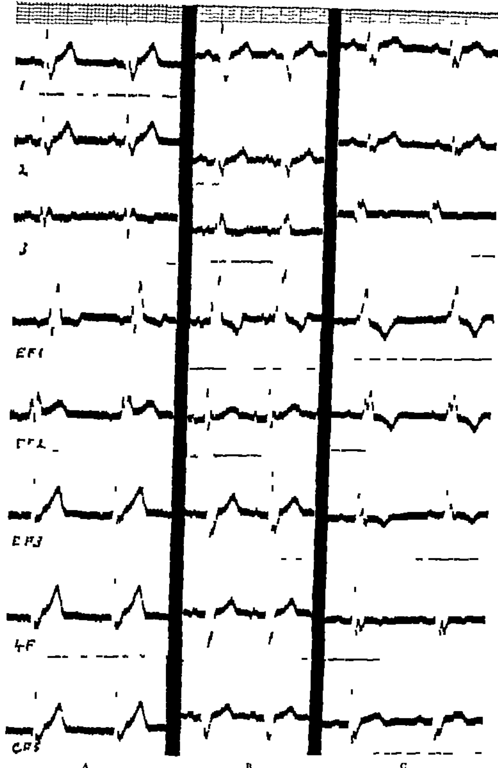


Fig. 79 Records indicating right bundle branch block. The three upper rows are the limb leads and below these precordial Leads EF 1, EF 2, EF 3, 4F and CF 5.

A Bayley's type 3, B type 2, C type 1.

It will be noticed that the peak of the R wave appears late in Leads EF 1 and EF 2, occurring 0.08 second after the beginning of QRS except in record B which shows the delayed R only in Lead EF 1. Leads from points farther to the left show an early apex of R 0.02 to 0.04 second after the beginning of QRS.

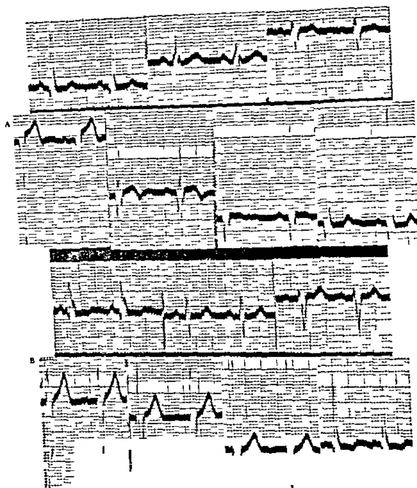


Fig. 31. Illustrating slight delay in the time of appearance of the peak of R in the precordial leads of records with marked left axis deviation and a QRS group of 0.1? second duration. The three limb leads are above and Leads EF 2, EF 3, 4 F and CF 5 are below in this order from left to right.

A. From a patient with long standing hypertension and enlarged heart. QRS duration equals 0.1? second; left axis deviation definite (+ + –). The R wave is absent in Lead EF ? and appears 0.06 second after the beginning of the QRS group in the other three precordial leads.

B. From another patient with hypertension and enlarged heart. The duration of QRS equals 0.1? second. Left axis deviation *marked* (+ 0 –). The peak of R is 0.03 second after the beginning of QRS *in Lead EF 2* and 0.0? second after the beginning of QRS in the other leads. T is inverted in Lead CF 5.

Lead I, taken simultaneously. In Leads IV F and CF 5 of normal hearts the peak of R may follow the beginning of the QRS of these leads by 0.04 second but the delay never reaches such a figure as 0.08 second as is found with left bundle branch block. Records with right axis deviation of QRS often have the peak of *R in Lead CF 2 as late as 0.04 second after the beginning of QRS* in this lead, but it never is found as late as 0.08 second after the beginning of QRS as occurs in records with right bundle branch block. These measurements therefore may afford a method of distinguishing between curves due to bundle branch block and marked right or left axis deviation of QRS due to preponderant hypertrophy of one ventricle.

The increased duration of QRS is so closely related to the mechanism of bundle branch block that the author believes that in order to be certain of the condition the QRS duration must measure more than 0.12 second. In 1914 Carter laid down criteria for the diagnosis of bundle branch block which included a widening of the QRS group beyond 0.10 second. He and many subsequent authors have considered that a QRS of 0.12 second is an adequate basis for a diagnosis of bundle branch block but there are actually numerous records which show this duration of QRS and which have none of the other characteristics of bundle branch block. The precordial leads of a number of these have been examined and have not been found to show the expected delay in the appearance of the peak of R on one or on the other side of the precordium (Fig. 31). Furthermore a duration of 0.12 second is an occasional though rare finding in records from normal hearts. In view of the above facts it does not seem sound to use the figure of 0.12 second as a criterion for the duration of QRS in bundle branch block. In order to afford a reasonably certain diagnosis its duration should exceed 0.12 second but the precordial leads should always be used to obtain the final decision, being guided by the delayed appearance of the peak of R either in the sternal positions (right branch block) or at the apex and axillary (left branch block) and by the fact that this peak is delayed as much as 0.08 or 0.10 second in the presence of block of either branch.

Curves such as those of Figures 22 B and 31 A and B are not

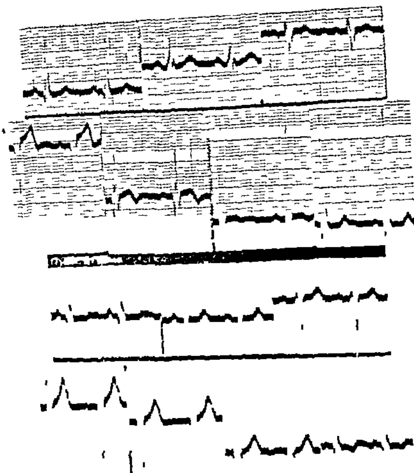


Fig. 3. Illustrating slight delay in the time of appearance of the peak of R in the precordial leads of records with marked left axis deviation and a QRS group of 0.1 second duration. The three limb leads are above and Leads EF 2, EF 3, EF 4 and CF are below in this order from left to right.

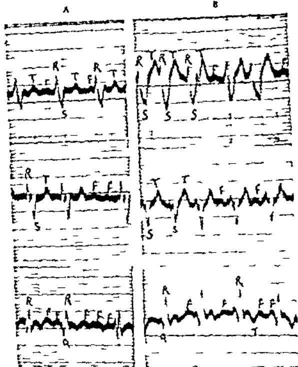
A. From a patient with long standing hypertension and enlarged heart. QRS duration equals 0.1 second; left axis deviation definite (+ + −). The R wave is absent in Lead EF 2 and appears 0.06 second after the beginning of the QRS group in the other three precordial leads.

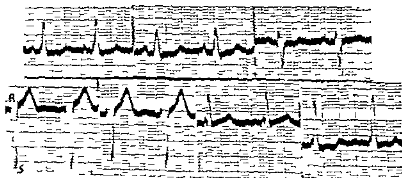
B. From another patient with hypertension and enlarged heart. The duration of QRS equals 0.1 second. Left axis deviation marked (+ 0 −). The peak of R is 0.03 second after the beginning of QRS in Lead EF 2 and 0.0[illegible] second after the beginning of QRS in the other leads. T is inverted in Lead CF 5.

uncommon and it is believed that when a record shows such a marked left axis deviation, that Lead 2 has a deflection approximately zero or definitely downward (Fig 22 B), then a duration of 0.12 second for the QRS group may often be due to the increased thickness of the ventricular muscle which must be traversed by the contraction (Chapter III) A complete bundle branch lesion produces a still greater widening of the base of QRS the figure reaching 0.14 second or more as has been said

It has been suggested that curves with a QRS duration of 0.12 second and with marked left axis deviation may have these abnormal features because of a delay in the spreading of the contraction due to a partial dysfunction of the left bundle branch This has been called *incomplete bundle branch block* The stimulus would thus be delayed in transmission to the left ventricle in a manner analogous to the delay in auriculoventricular conduction shown by a prolonged P R interval Yater has studied one such case histologically and found fibrosis of the left bundle branch without marked disturbance of its continuity He considered that this fibrosis was the reason for the bundle branch having a depressed function so that the conduction through it was delayed This sort of pathological process may explain the occasional cases developing left bundle branch block after having previously shown marked left axis deviation of QRS with inverted T_1 Certain records with marked left axis deviation of QRS a QRS duration of 0.12 second and an inverted T_1 have been found by the author to have the peak of the R wave of *Lead CF 5 delayed until 0.06 second after the beginning of QRS* Such records probably indicate incomplete left bundle branch block (Figs 31 A and 32 C)

The diagnosis of right ventricular hypertrophy versus incomplete right bundle branch block is less difficult, for the right *ventricle does not increase in thickness to the same extent as the* left In fact I have not seen a record with right axis deviation with a QRS duration of 0.12 second except where notching was also present indicating a probable lesion of the Purkinje fibers Figure 32 A might perhaps have been considered a record with a doubtful significance for it has a QRS duration of 0.12 second an atypical notched QRS group of the right axis deviation type





C

F 5 Illustrating incomplete bundle branch block

A Auricular fibrillation with incomplete right bundle branch block. The QRS duration equals 0 1 second and the notching and unusual appearance of the QRS group indicates a disturbance of the spreading of the contraction similar to that of right bundle branch block

B A later record of the same patient showing complete right bundle branch block type I with a QRS duration of 0 14 second or greater

c. Normal rhythm with incomplete left bundle branch block. The limb leads and precordial leads are as in Figure 31. The QRS duration equals 0 1? second. The appearance of QRS in the limb leads is not suggestive of the usual curves of left bundle branch block but the precordial leads show a late R peak 0 07 second after the beginning of QRS in Leads 4 F and CF 5. Note the tiny R preceding S in Lead EF 9

and a T wave which is always opposite in direction to the chief deflection of QRS The patient had a mitral stenosis of long standing and a much hypertrophied heart This record is to be diagnosed as indicating a delayed conduction (incomplete block) in the right bundle branch because of the long duration of the QRS group combined with notching and the broad S in Lead I The diagnosis was confirmed by the later development of complete obstruction of the right bundle branch during a period of acute cardiac failure, when Figure 32 B was obtained This has a QRS duration of 0 11 second and the complex is typical of a right bundle branch lesion with a Type I complex (page 118)

It would seem then that incomplete bundle branch block is manifested by a prolongation of the duration of QRS and changes in the waves of the QRS group analogous to those attributed to preponderant ventricular hypertrophy of the affected side The more characteristically the QRS and T waves approach the configuration expected with complete bundle branch block and the wider the duration of QRS the more reason there should be to diagnose incomplete bundle branch block rather than preponderant ventricular hypertrophy Figure 32 C is a curve that is probably the result of incomplete left bundle branch block It has a QRS duration of 0 12 second with R the chief QRS deflection in Lead I and S in Lead 3 T_1 is upward an unusual but not unheard of finding in left bundle branch block The precordial leads are similar to those found with left bundle branch block in having a small or absent R in leads from the sternal and parasternal positions and a delayed R at the apex and further left (Fig 30) In this record the peak of R in Lead 4 F is 0 07 second after the beginning of QRS in that lead a lesser delay than is usual with left bundle branch block (Fig 30) but more than is usual without it

In certain patients bundle branch block complexes are constantly present in others they are transient When the fundamental tendency is present they may sometimes be induced by quinidine by exercise by deep inspiration and by other influences The phenomenon of alternating bundle branch block has been observed in certain cases which showed alternating cycles with a normal and with a bundle branch block configuration

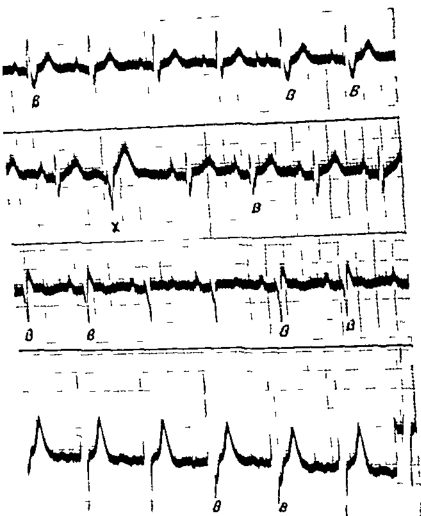


Fig 33 Intermittent bundle branch block Leads 1 2 3 and 4 F The complexes marked B in each lead indicate right bundle branch block The other complexes are of a more normal form except for the one marked X in Lead 2 which is a ventricular ectopic beat The bundle branch complexes are of Bayley's type 3

and a T wave which is always opposite in direction to the chief deflection of QRS The patient had a mitral stenosis of long standing and a much hypertrophied heart This record is to be diagnosed as indicating a delayed conduction (incomplete block) in the right bundle branch because of the long duration of the QRS group combined with notching and the broad S in Lead I *The diagnosis was confirmed by the later development of com* plete obstruction of the right bundle branch during a period of acute cardiac failure when Figure 32 B was obtained This has a QRS duration of 0 14 second and the complex is typical of a right bundle branch lesion with a Type I complex (page 118)

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In certain patients bundle branch block complexes are con stantly present in others they are transient When the funda mental tendency is present they may sometimes be induced by quinidine by exercise, by deep inspiration and by other influ ences The phenomenon of alternating bundle branch block has been observed in certain cases which showed alternating cycles with a normal and with a bundle branch block configuration

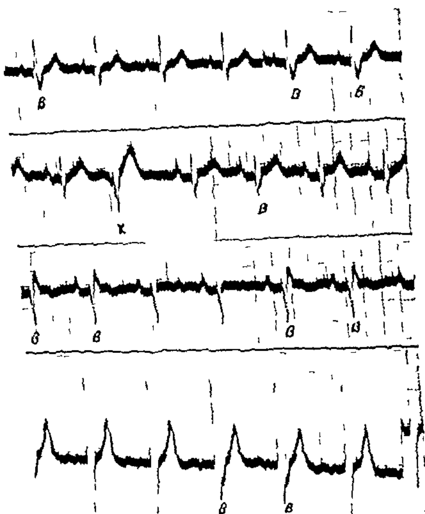


Fig. 33. Intermittent bundle branch block. *Leads 1, 2, 3 and 4.* The complexes marked *B* in each lead indicate right bundle branch block. The other complexes are [illegible] normal [illegible] *except for* the one marked X in Lead 2 which is a ventricular ectopic beat. The bundle branch *complexes* are of Bayley's type 3.

Other records, like Figure 33 have shown bundle branch block complexes occurring irregularly in a sequence with normal forms Sometimes with arrhythmia the more normal ventricular complexes only appear after a long diastolic pause

A partial depression of conduction in one bundle branch might be expected to occasionally produce a record with complexes gradually changing from an approximately normal form to one typical of bundle branch block the mechanism of gradually increasing conduction time in the bundle branch being analogous to the Wenckebach phenomenon observed with partial depression of A V conduction Such records in fact have been observed and Sherf has published in his book (page 63) an example of a transition in three heart cycles from a bundle branch block complex to one with normal QRS duration

There is a particularly interesting group of cases first described by Wolff Parkinson and White which usually show complexes typical of left bundle branch block a short P R interval (auriculoventricular conduction time approximately 0 08 second) and a tendency to attacks of paroxysmal tachycardia Such a record is seen in Figure 34 Two cases have been reported by Tung with short P R interval and the ventricular complexes of right bundle branch block The bundle branch block is not always a constant phenomenon in the individual case but may alternate with periods of relatively normal ventricular complexes One case has been reported by Mora and Inchauspe showing the short P R with bundle branch block complex alternating regularly or giving place irregularly to beats with a P R interval of 0 16 second and QRS duration of 0 08 second Various hypotheses have been brought forward to explain this phenomenon but the most acceptable one seems to be that suggested by Cossio who considers that the complexes with short P R interval and bundle branch block are due to the excitation of an overirritable focus in the ventricle by the auricular systole The possibility of conduction of the impulse through a short accessory pathway (auriculoventricular pathway) known as the Kent bundle might explain those cases with curves resembling those of left bundle branch block but does not explain the occurrence of curves of the type of right bundle branch block

Before discussing those electrocardiographic changes which are due to a less well defined pathological basis it may be well to consider separately the effects which may be produced upon the

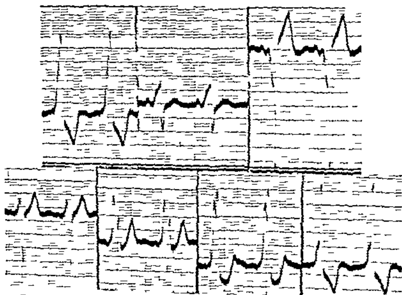


Fig 34 Bundle branch block with short P R Leads as in Figure 31 The amplitude of the excursions in this record is unusually great The first and fourth *precordial leads are standardized* at one half standardization 5 mm = 1 millivolt The P R interval is 0 10 second in the limb leads

ventricular waves by the various influences which might affect the myocardium in the course of cardiac disease This is intended to be a schematic presentation and it must be borne in mind that the end result when two *or more of* these influences are present simultaneously will be a balance of the effects of each The effects of damage to the main bundle branches *are* always so predominant that when such damage is present the curve will register this in spite of all other influences that may be affecting the muscle

A Overactivity or hypertrophy

QRS voltage may be increased

QRS duration may be increased (only by marked hypertrophy of left ventricle)

T voltage may be increased

T may be inverted in Lead I (only with marked hypertrophy of left ventricle)

B Diffuse disease or depression of function

QRS voltage may be decreased

QRS duration may be increased

Q T duration may be increased

T voltage may be decreased

C Localized disease of ventricular muscle

a Not affecting large subdivisions of the bundle branches

QRS voltage may be increased or decreased

QRS may be notched

QRS individual peaks and direction of axis may be changed if affected myocardial area is large

T voltage may be increased or decreased

T may be abnormally inverted

b Affecting large subdivisions of bundle branches

QRS voltage may be increased

QRS direction of axis may be changed individual peaks may be changed

QRS duration may be increased

QRS may be notched

c Affecting a main bundle branch

The curves characteristic of bundle branch block will appear

In discussing the abnormalities of the electrocardiogram which may result from disease the first step will be to define the abnormal characteristics of a wave and then to indicate the varieties *of disease which may cause these features*

Notching of the QRS group It has been explained in Chapter II that notching or slurring of QRS may appear in one lead *of a normal record and even in two leads provided that it does* not appear in either of them upon a part of QRS which is near the top of a tall peak When notching or slurring appears in more *than one lead near the top of a tall peak it should be considered* to indicate an abnormality of the record Such notching is usually though not always accompanied by an abnormal inver

sion of the T wave If the notching is slight and occurs during a quickly moving excursion it may be straightened out as it were into a sort of thickening or slurring of the side of an R or S wave Notching is illustrated in Figures 5, A and C 3, A and B and 27 A and B while slurring can be seen in Figure 3, A B and C and in many other records

It is usually evident that the notching of QRS occurs at the same time interval after the beginning of the QRS group in one lead as it does in another or perhaps notching in one lead will be found at the same time as slurring in another This is because these notchings or slurrings in the different leads are due to the same disturbance in the production of the electrical potential within the heart This disturbance of potential may arise (1) from the failure of a diseased area of the myocardium to contribute its electrical potential so that the normal development of the curve is interrupted or (2) from an interference with the normal spreading of the contraction wave by a lesion which affects a large area of the Purkinje ramifications This prevents the myocardial areas supplied by these ramifications from receiving the stimulus normally and entering into activity at the normal time It must receive the stimulus from the nearby ventricular muscle and therefore its potential is delayed

Lesions affecting only small areas of the Purkinje ramifications probably do not lead to the occurrence of notching or slurring for the contraction is spread around and past them by the intact branches at a considerable speed It has been shown by experimental work that such lesions as are caused by a knife-cut across the endocardial surface of the ventricle of the dog do not lead to appreciable changes in the ventricular waves and particularly not to notching of the QRS group unless the main bundle branches are involved

If notching is accompanied by an increase in the duration of the QRS group it is probably due to a lesion of the Purkinje system as in Figure 32 A If notching is present without an increased duration of QRS it may be due to a focal diseased area in the ventricular muscle without interruption of main Purkinje tracts The notching in Figure 3, A and B probably belongs in this category Price and Pardee however on careful histological

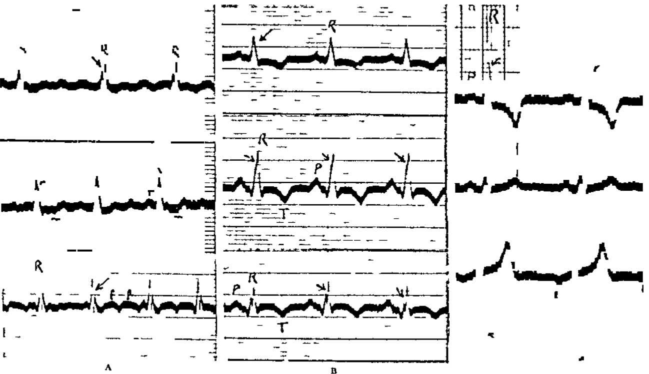


FIG 35 To illustrate notching or slurring of the QRS group This occurs at the points indicated by the arrows

A shows notching of R 1 and R 3 and slurring of R 2 Auricular fibrillation is present and the T waves are so small in amplitude as to be scarcely discernible

B shows slurring of R 1 and R 2 and notching of R 3 There is an abnormal inverted coronary T wave in Leads 1 and 2 and also a coronary T in Lead 3

C shows marked left axis deviation of QRS with slurring of R 1 and R 3 and an abnormal inverted T wave in Lead 1 The duration of QRS is 0 12 second The S T segment is below the isoelectric level in Lead 1 and above it in Lead 3

study of six hearts whose records showed abnormal notching and slurring of QRS as *the only abnormality of the curve* found that each one had diffuse myocardial disease without large focal areas These cases prove that not only large focal areas but also diffuse disease may cause notching and slurring of QRS

A notched QRS is very likely to have associated a downward T_1 or T especially if the duration of QRS is increased The abnormal inversion of T in these records may result either from the change in the time relations of the right and left ventricular contractions due to a lesion of the Purkinje system or from the abnormal electrical production resulting from a large area of myocardial disease

In precordial leads notching and slurring of the QRS group is not abnormal in records obtained from the region of the interventricular septum and cardiac apex or from the midline and to the right of this In other regions it may be a manifestation of myocardial disease beneath the site of the electrode for example Lead EF_2 of Figure 31 A It has been pointed out by Wilson that in direct leads from the heart muscle it is commonly found at the margin of an area of infarction

Large Q waves It has been observed that many electrocardiograms from patients with coronary arteriosclerosis show an unusually large Q wave in the third lead * This was mentioned in the first edition of this book and has been noted by numerous authors since then The chief difficulty in using this as a basis for diagnosis lay in deciding when the Q wave should be considered abnormally large After a review of a large number of records of both normal and abnormal hearts it seemed to the author that Q_3 should be considered abnormally large when it measured 25 per cent or more of the largest peak of QRS found in any of the limb leads (Fig 36) If however right axis deviation was present or if the R wave was followed by an S it was not believed that the large Q_3 had the same significance Likewise the M type of complex with two upward peaks was excluded as the downward peak was not called Q The practical value of

* Records indicating bundle branch block are to be excluded from this consideration of the Q wave

this measurement has been confirmed by subsequent investigators although Durant and Peel have indicated that in many cases the M type of complex like that of Figure 92 c may have

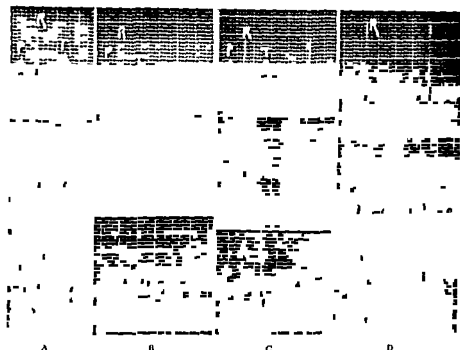


Fig 36 To illustrate the large Q 3 In each record there is left axis deviation of the QRS group and in each record the negative deflection in Lead 3 due to the difference between the height of R 1 and R 2 must be found in the Q wave In all records this Q wave has a value of more than 25 per cent of the height of the largest peak in the limb leads Note the low voltage T wave of records B and D also the diphasic (— +) T in Leads 1 and 2 of record C

Note especially the second heart cycle in Lead 3 of record B The first movement of this QRS group is upward followed by a downward wave and then an upward wave This is the type of complex which has been referred to as the M type This was only an occasional occurrence in the third lead of this patient the majority of the complexes being like the first one here shown and having no upward deflection preceding the downward wave marked Q

a similar significance to the large Q_3 and likewise the downward deflection QS without any upward deflection

Kossmann suggested after a study of the size of Q in a series of normal hearts that if Q_3 is more than 25 per cent of the largest peak of QRS in this lead it is probably abnormal He also confirmed the author s standard of 25 per cent of the largest peak of QRS found in the limb leads Wilson s table (page 12) indicates 3 5 mm as the maximum normal value When Q_3 is large there is often a Q in Lead 2 also but in many cases there is not

Peel found a greater frequency of cardiac disease in patients with large Q_3 having also a Q though Q need not in such cases be large He stated that a Q_3 of 6 mm was to be considered abnormal in any case

Kossmann suggested that when Q exceeds 20 per cent of the largest peak of QRS in Lead 2 it is probably abnormal while Wilson s figures indicate 3 25 mm as the upper limit of normal for Q Peel found that a Q of 3 mm or more was an indication of myocardial disease

It is likely that the appearance of even a very small Q wave in Lead 1 may occasionally be an abnormal feature and especially so in the presence of right axis deviation of QRS The only suggestions as to normal criteria for Q_1 are derived first from Kossmann s conclusion that a value exceeding 15 per cent of the largest peak of QRS in this lead should be considered abnormal and second from the figure in Wilson s table (page 12) which suggests that a measurement of 2 46 mm would be abnormal in a record from a young adult

Durant set up certain standards for abnormality of the Q wave and found that the larger the value of Q_3 in relation to *that of the* largest peak of QRS and the larger the Q associated with it the greater the likelihood of coronary arteriosclerosis being present He found however that numerous definite coronary cases were eliminated by making the criteria more exacting He also set up certain criteria for Q_1 which were somewhat less definitely associated with coronary disease than his criteria for Q_3 In this study we encounter a different terminology from that which others have used in the description of the Q wave for he described by this name those QRS groups without an R wave in which the sole deflection of QRS was downward (the QS deflection) When such curves were eliminated from the Q_1 group the frequency of coronary arteriosclerosis was found definitely greater in the remaining cases thereby showing that calling such waves Q leads to a grouping together of curves of different significance

An abnormally large Q_1 is sometimes associated with an inverted T wave in this lead and a large Q_3 may be associated with inversion of T_3 and possibly of T_2 also Such association increases

the probability of coronary arteriosclerosis but the lack of T wave inversion does not detract from the diagnostic importance of the large Q wave

The large Q_3 is much more frequent than a large Q wave in the other leads and has many important associations It is not known however to what structural changes this abnormality of Q_3 should be referred It is an interesting fact that it is found not only in records from patients with coronary arteriosclerosis especially those with the anginal syndrome but also in records from patients with rheumatic pericarditis and occasionally with acute rheumatic fever without evidences of pericarditis It has been observed after pulmonary embolism and in infestation with trichina

It was observed that variations in the position of the heart due to movements of the diaphragm have a profound effect upon Q_3 in many cases It may be large in the normal position of the diaphragm and a full inspiration may diminish its size or may even entirely abolish it This has been observed in cases known to have coronary arteriosclerosis Our present knowledge does not indicate that the disappearance of the Q wave on deep inspiration places it in any different category from that of a Q wave which is not abolished by deep inspiration Clinical observations concerning this wave have only been made with the diaphragm *in its normal respiratory position The influence of respiration* upon it has no diagnostic significance at present

In precordial leads the Q wave is not normally found in records from positions *1* and *2* and only occasionally from position *3* If it exceeds *3* mm in Lead *4 F* or *3 5* mm in Lead *CF 5* it is probably abnormally large In records after infarction of the anterior surface of the heart it is usual to find large Q waves in the leads from the apex and beyond and in Lead *EF 3* (and *CF 3*) it is usual to find large downward deflections (QS wave) without any R wave either preceding or following These waves have been called large Q waves by Wilson and his followers Similar waves are frequently found in Lead *EF 2* (and *CF 2*) The absence of the R wave in these precordial leads has been attributed to the absence of the muscular contraction over the area of the infarction The large downward deflection—QS wave—is then

the result of the activity of the septal muscle opposite to the infarcted area The large Q wave and small R which appear in precordial leads obtained from points further to the left are considered as evidence of areas of degenerated myocardium combined with areas of undegenerated tissue

The duration of Q_3 The significance of an increased duration of Q_3 has been investigated by Bayley who found a high frequency of coronary arteriosclerosis among patients showing a Q_3 with a duration of 0.04 second or more All curves showing right axis deviation or having QRS complexes with a duration of 0.11 second or more or having M or W shaped complexes in Lead 3 were excluded from consideration He included both Q waves and QS waves in his group He found that the frequency of coronary disease was greater when there was also a Q even though this was of small size It is interesting that in addition to coronary disease these wide Q waves occurred in patients with rheumatic heart disease and in his group of 163 patients there was one patient with myxedema one with neurocirculatory asthenia one with anemia and one normal pregnant woman In spite of the inclusion in this group of a few patients without cardiac disease the finding of an electrocardiogram with the duration of Q_3 .04 second or more seems to have definite diagnostic value

Duration of the QRS group Increased duration of the QRS group indicates a delay in the spreading of the contraction throughout the ventricular *muscle* As has been pointed out this may depend upon an increased thickness of the muscle of the left ventricle or upon a dysfunction of the system of fibers which distributes the contraction stimulus to the ventricular muscle i e the right and left branches *of the* A V bundle and the Purkinje fibers within either ventricle If the stimulus is spread uniformly but slowly throughout the *ventricles* there will be a prolongation of the QRS group—an increase in its width without any change in its form A prolong*ation of QRS* occurs for this reason in some patients whose circulation has seriously failed and in some whose heart rate has greatly increased An increase is observed after the administration of quinidine which delays the rate of propagation of the impulse (*Fig* 78) Figure 32 A and B

are records obtained at different times from the same patient showing an increased duration of the QRS group associated with increased heart rate in a heart already so affected by disease as to have incomplete right bundle branch block

Localized disease affecting a considerable area of the Purkinje network or a large subdivision of the right or left bundle branch may lead to a prolongation of QRS because the area which should receive its stimulus by way of the diseased tissue is late in contracting The amount of prolongation resulting from this would depend upon the size and situation of the area of ventricular muscle activated by the diseased conducting tissue Such a pathology would very likely lead to notching of QRS as well as a prolonged duration because of the interference with the order of the contraction

It should be mentioned again here that Wilson and Herrmann after a correlation of the duration of QRS and the size of the heart concluded that a QRS duration of over 0 10 second can not always be ascribed to an increase in the thickness of the ventricular wall They feel that it is an indication of delayed intraventricular conduction

Pardee and Price found that considering the QRS duration in a group of records with left axis deviation and excluding those diagnosed as bundle branch block the average weight of fifteen hearts with normal QRS duration was 185 gm while the average heart weight of eleven hearts with increased duration was 560 gm This suggests a relation between heart size and QRS duration but there were two large hearts one 680 gm and one 800 gm with a normal duration of QRS and one small heart 275 gm with increased duration Cardiac enlargement evidently need not cause QRS prolongation and evidently is not its sole cause One must of course consider the effect of marked muscular thickening of the left ventricle which would prolong the spreading of the contraction more than it would contribute to an increase in heart size In a given case the precordial leads will give information about the time of arrival of the stimulus in either ventricle which should enable us to decide whether or not there is delayed conduction in one bundle branch (page 128)

A shortening of the duration of the QRS group is expected

with increased rate but it is found under a few other circumstances In records from children QRS may have a duration of only 0 06 second possibly because of the small size of the ventricles so that the spreading is quickly completed A very brief QRS may be found in the record from a heart which has just suffered from an infarction which has destroyed the function of a considerable area of muscle (Fig 63 A)

In precordial leads the duration of QRS is in general of the same order of magnitude as is found in the standard leads of the same record It has been observed however that when the standard leads show a slightly prolonged duration this is often longer in the precordial leads by perhaps 0 01 or 0 02 second

Voltage of the QRS group The height of the largest wave of the QRS group of the limb leads seems to vary for much the same reasons as does the height of P It is usually large when there is an increased amount of muscle whether or not the hypertrophy leads to a preponderance of one ventricle It is larger when the physiological state of the muscle is better and smaller when this is not so good The upper limit of normal has been set at 20 mm and the lower at 5 mm but with waves as large as QRS it is always necessary to add 15 per cent to the largest recorded value when one lead has a small relative excursion This has been discussed in Chapter II where it was pointed out that under these conditions the minimum recorded value of QRS might be as little as 4 4 mm and the maximum should not be over 17 4 mm

After the acute infectious diseases and during marked circulatory failure the voltage of QRS is often found small It increases during convalescence and with improvement of the circulation Figure 37 A and B are records from the same patient the first taken just as he was recovering from a period of severe cardiac failure and the second three weeks later after he had improved greatly under treatment Note the increased voltage of QRS and also of T in the second record

Many patients with sclerosis and narrowing of the coronary vessels give records with the QRS excursions of very small size and these often do not increase to normal size even when com

pensation improves. Figures 38 A and B and 28 B are from such patients. Record 38 B shows an interesting feature in the small excursions of the waves of the ventricular extrasystoles showing



Fig. 37. Two records of the same patient, the first taken just after recovery from severe heart failure and the second several weeks later after he had further regained compensation. Both records show auricular fibrillation, both show marked left axis deviation of QRS and both show a downward T wave in Leads I and 2. The difference lies in the voltage of the QRS group and of the T wave, record B showing the larger voltage of both QRS and T.

that with an abnormal cardiac contraction as well as with the normal the myocardium did not produce the usual electrical potential.

We are not certain of the cause of low voltage of QRS but it can perhaps be best stated as being something concerned with a depressed physiological condition of the muscle, sometimes dependent upon deficient nutrition as with coronary arteriosclerosis or with wasting diseases such as carcinomatosis, sometimes having a metabolic origin as with hypothyroidism. It is often found during lobar pneumonia, possibly an indication of a toxic effect upon the myocardium. It is occasionally found with an acute rheumatic myocarditis where the inflammatory reaction is

probably the cause It is also found with severe anemia pulmonary tuberculosis and bronchial asthma

In addition to this fundamental myocardial cause for low volt



Fig 59 Records showing low voltage of QRS and low voltage of T [illegible]
the voltage of P [illegible]
ventricular [illegible]
excursion [illegible]

age QRS there is an extracardiac factor which is probably due to a short circuiting of the electrical potential before it reaches the extremities Pericardial effusion may cause low voltage by this mechanism or possibly by the pressure upon the coronary veins slowing the blood flow and leading to poor myocardial nutrition Emphysema is often associated with low voltage of QRS and this is possibly due to short-circuiting of the heart s current by the overlapping lung though Katz s suggestion that the lung is a poor electrical conductor must be considered

A word of caution is necessary against confusing the phrase poor physiological condition which has been used here to describe the state of the heart muscle that leads to a low voltage in the limb leads with any idea of the heart s ability as a pump

or with the degree of cardiac functional capacity which might be expected The latter are dependent upon many factors other than this particular feature of the myocardium and it is an



Fig 39 A The downward T wave in Leads 2 and 3 which frequently appears in records with right axis deviation of QRS

B Showing normal electrical axis of QRS with the T wave turned downward in all three leads This patient was not under the influence of digitalis The QRS group is abnormally wide measuring 0 12 second in Lead 3

C *A heart with congenital abnormality showing the unusually large excursions* of QRS along with a notching and increased duration of QRS that is rare in such records

observed fact that many patients with low voltage of QRS do not show evidence of cardiac insufficiency or the anginal syndrome and in fact may not show any cardiac symptoms or abnormal signs Turner in reviewing 164 records with low voltage of QRS found congestive failure in 70 cases but no congestive failure in 43 other cases with definitely diseased hearts In patients without heart disease an abnormal amount of tissue fluid was present in 39 but was absent in 12 cases

Most patients with congenital cardiac abnormalities have very large *excursions* of QRS (Fig 39 C) also many with aortic regurgitation or marked mitral stenosis (Fig 2[illegible] A) or high blood

pressure (Fig 22 B) In all these conditions the cardiac hypertrophy is probably the factor which causes the increased size of the deflections The size of QRS will always be a resultant of several varying influences A heart whose R wave would be very large if the muscle were in good condition may show one within normal limits or even less if the physiological condition of the muscle is below par or if there is short-circuiting of the heart's current by fluid or by emphysema

Should the state of nutrition of the heart muscle vary from one time to another it will lead to variations of the voltage of QRS no matter whether the waves are normal notched or of the type associated with bundle branch lesions Compare Figures 24 A and 28 A two records of the same heart the first taken when the patient was not well compensated the second at a later period when the condition had become serious Variations in the height of the waves can only be attributed to variations in the nutritional state of the muscle when the form of QRS remains the same showing that the contraction is spreading by the same path each time for it has been pointed out that variations in the path of the contraction can cause variations in the height of the waves

It is not possible to attach importance to slight differences in the height of the waves of records from different persons—to affirm that the muscle of one heart is in better or in worse condition than that of another There are too many independent factors which can increase or decrease the voltage for example the degree of hypertrophy the path of the contraction the physiological condition of the muscle fibers or factors in the surrounding tissues which might influence the short-circuiting of the heart's current

The *precordial leads* of hearts with low voltage of QRS in the standard leads usually show an amplitude of the QRS deflections within the range of normal though they may be low in certain leads Figure 40 A shows a record from a patient with old healed cardiac infarcts and with good ability to carry on the ordinary activities of life The amplitude of QRS [illegible]

[illegible] adjacent part of the myocardium

or with the degree of cardiac functional capacity which might be expected. The latter are dependent upon many factors other than this particular feature of the myocardium and it is an

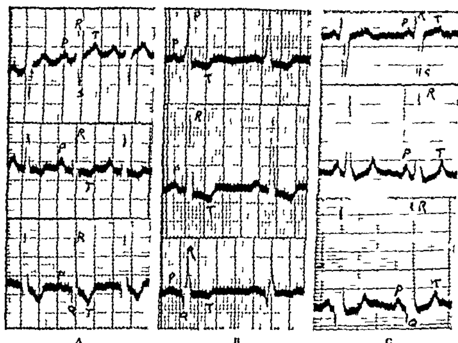


Fig. 39. A. The downward T wave in Leads 2 and 3 which frequently appears in records with right axis deviation of QRS.

B. Showing normal electrical axis of QRS with the T wave turned downward in all three leads. This patient was not under the influence of digitalis. The QRS group is abnormally wide, measuring 0.12 second in Lead 3.

C. A heart with congenital abnormality showing the unusually large excursions of QRS along with a notching and increased duration of QRS that is rare in such records.

observed fact that many patients with low voltage of QRS do not show evidence of cardiac insufficiency or the anginal syndrome and in fact may not show any cardiac symptoms or abnormal signs. Turner in reviewing 164 records with low voltage of QRS found congestive failure in 70 cases but no congestive failure in 43 other cases with definitely diseased hearts. In patients without heart disease an abnormal amount of tissue fluid was present in 39 but was absent in 12 cases.

Most patients with congenital cardiac abnormalities have very large excursions of QRS (Fig. 39 C), also many with aortic regurgitation or marked mitral stenosis (Fig. 22 A) or high blood

obtained over the right ventricle from positions 2 and 3 show an unusually large S wave In many cases the S wave will be unusually large with all positions of the electrode When the left ventricle is hypertrophied curves obtained at the apex and beyond show an unusually large R wave and in some cases the R is unusually large from positions 2 and 3 Occasionally it may be small in records from these positions or in position 2 may be absent It is doubtful whether this latter is an effect of uncomplicated hypertrophy or whether it may not be due to a complicating myocardial disease

Voltage of T In general myocardial conditions which affect the voltage of QRS will affect the voltage of T in a similar manner so that they tend to be increased or decreased at the same time Certain poorly understood factors however may influence only the T wave or may affect it more than QRS is affected so that in certain records T shows the chief change

The voltage of T is changed when the ventricular myocardium is affected by any one of the three following factors (1) the physiological state of the myocardium (2) the presence of organic myocardial disease and (3) an abnormal spreading of the contraction When the physiological state of the myocardium is normal T will tend to have a normal voltage When the myocardial nutrition is depressed the voltage of T will be diminished When the strength of the ventricular contraction is abnormally increased T will tend to be large It tends to be larger after exercise and during acute thyrotoxic states and with ventricular hypertrophy Although pathological changes in the myocardium can profoundly affect the voltage of T it is difficult or impossible to lay down general rules as to whether the voltage will be increased or decreased More extensive lesions tend to be associated with a low voltage of T because they are usually due to coronary narrowing or acute inflammation or toxemia and result in a depressed physiological state of the muscle Master found that coronary arteriosclerosis acute rheumatic myocarditis ch[illegible] valvular disease [illegible]
the most frequen[illegible]
low voltage T If [illegible] to involve one of the bundle branches or a main subdivision the spreading of the contraction

Figure 40 B is the record of a patient who had a slightly abnormal tendency to shortness of breath and transient attacks of auricular fibrillation The amplitude of the R and S waves of all precor

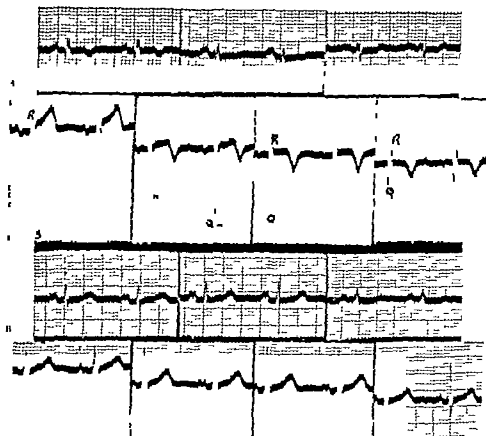


Fig 40 Records with low voltage of QRS in the limb leads The upper row shows Leads 1 2 and 3 The lower row shows Leads EF 2 IF 3 4 F and CF 5

A From a patient with healed anterior infarction This shows the Q 1 T 1 type of ventricular complex absence of R in Lead FI 3 a tiny R in Lead 4 F and a small R *in Lead CF 5 The T wave in the precordial leads as well as in the* limb leads is such as is associated with a healing anterior infarction

B From a patient with generalized coronary arteriosclerosis and mild cardiac insufficiency The R S and T waves of the precordial leads are normal although the limb leads show a low voltage of QRS

dial leads is within the normal range though the voltage of QRS in the limb leads is low

The precordial leads of records from hearts which are known to have hypertrophied musculature usually show large total QRS excursions (R + S) With right ventricular hypertrophy curves

obtained over the right ventricle from positions 2 and 3 show an unusually large S wave In many cases the S wave will be unusually large with all positions of the electrode When the left ventricle is hypertrophied curves obtained at the apex and beyond show an unusually large R wave and in some cases the R is unusually large from positions 2 and 3 Occasionally it may be small in records from these positions or in position 2 may be absent It is doubtful whether this latter is an effect of uncomplicated hypertrophy or whether it may not be due to a complicating myocardial disease

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may be so disturbed as to lead to an increase in the voltage of T This is why a large voltage of T is often found in records with a notching of the QRS group When low voltage of T is found in a record with notched QRS it will probably be the result of a diffuse depression of myocardial function by diffuse disease

To state a hypothetical comparison of the influence of these different factors upon QRS and upon T a heart without pathological processes in the ventricular muscle but with the muscle in a poor physiological condition such as might result from lack of exercise because of a sedentary life or from severe anemia might give a low voltage QRS and a low voltage T With slight diffuse pathology in the ventricles such as might be due to arteriosclerotic narrowing of the coronary arteries (Fig 37 A) or to the toxemia of an acute disease the QRS voltage might be normal though small and the T wave less than the normal voltage The greater the involvement of the muscle the smaller would be the voltage of T unless a large division of a main bundle branch became involved in which case QRS would be changed and T might become of normal size or large

It is necessary to bear in mind the important distinction which has been made between low voltage of the T wave and a low amplitude of the T wave in any one lead Though the T wave may have less than 1 mm amplitude in any one lead yet if its amplitude in the other leads is found to be 3 or 4 or 5 mm this T wave is shown by the larger excursions to have a normal voltage While a low amplitude wave in one lead might indicate a transition stage between an upward and a downward deflection yet a low voltage T wave low in all leads does not indicate this

In precordial leads the amplitude of the T wave does not seem to parallel the voltage of T as recorded in the leads from the extremities In this respect the situation is similar to that of the QRS group Low voltage T may be accompanied by precordial leads with T waves of normal or large size and as is usual the amplitude of T may be found to vary in size in leads from different precordial points In certain records the T wave may be of small size in precordial leads from all the usual positions (Figs 41 and 77 A) The causes of this finding are not understood but it is probably related to the presence of an abnormal myocardium

as in these instances Large amplitude of T in the precordial leads is commonly found in records of hearts with left bundle branch block with hypertrophied musculature and also in certain

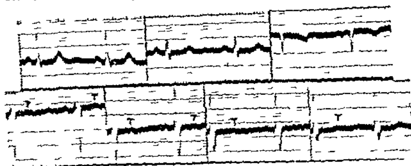


Fig [illegible] Record from a patient with adiposity and hypertension with symptoms of cardiac insufficiency There is a T wave with a normal voltage in the limb leads but in the precordial leads the amplitude of T is small with all positions of the electrode

phases of the process of healing following coronary thrombosis (Fig 69 B)

Abnormal direction of T The T wave should be directed upward in Lead 1 and in Lead 2 In about two thirds of the records from normal hearts it will be directed upward in Lead 3 also If it is found isoelectric or diphasic in either Lead 1 or Lead 2 it should be considered abnormal for such deflections represent a transition between an upward and a downward wave

The T wave like the QRS group has a normal and an abnormal direction for its chief electrical axis The normal T with upward deflections in Leads 1 and 2 and either an upward or a downward one in Lead 3 (+++ or ++−) has an axis which is seen from Figure [illegible] to lie between 0 and 90° The range for its normal direction is greater than for the QRS group When T gives a downward deflection in Lead 1 (−++) or Lead 2 (−−+) or (+−−) its electrical axis is abnormally directed Whether it is deflected to the right or the left of its normal position often seems to depend upon the direction of the electrical axis of QRS (page 101)

The conditions which change the direction of the electrical axis of T seem to be those associated with a prolongation of the contraction in a portion of the ventricular mass Possibly a local

ized shortening of the duration of contraction would also produce a change in the direction of T *It seems that a localized area of scarring also may have this effect* though the physiological process involved is not known

There is good experimental evidence to support the idea that abnormal inversion of T is caused by a prolonged activity of a part of the ventricular muscle Wilson and Finch have shown that the T wave of the human electrocardiogram can be changed in its direction by drinking a large amount of iced water This is ascribed to the cooling of the diaphragmatic surface of the heart which lies against the stomach The T wave of experimental animals is similarly affected when localized areas of the ventricles are cooled by the direct application of such cooling agents as the ethyl chloride spray or the carbon dioxide pencil *Cooling depresses the physiological activities of the affected area* prolonging the duration of its contraction and we have here a demonstration that such a prolongation can cause an abnormal inversion of T in an otherwise healthy heart

In the case of premature ventricular beats and of bundle branch lesions T is often directed opposite to the chief excursion of the QRS group in two leads if not in three, so that it frequently is inverted The abnormal inversion of T in these records is due to the delayed relaxation of the second ventricle to become involved *in the contraction* This prolonged activity of one ventricle tends to make T opposite to QRS and thus leads to its inversion This is especially true if the area of the QRS group rather than the size of the peak of largest amplitude is considered as indicating the direction of QRS according to the method suggested by Wilson (page 119)

When QRS shows abnormal notching or slurring it is common to find that T is downward in Lead 1 or Lead 2 or both (Fig 35 B and C) This change may be attributed to the effect of the basic pathology upon *the* character of the ventricular contraction *If* the notching is due to a Purkinje lesion the change in the direction of the T wave is ascribable to the delayed activity of the region of the ventricular muscle to which the affected branch is distributed If notching is due to a large myocardial focus the changed direction of T may arise because the diseased

area does not enter into contraction In the one case the abnormal T is due to an abnormal time relation of the events of the ventricular systole while in the other it is due to an abnormal (electrical) balance during the contraction

When left axis deviation of the QRS group is present it is not uncommon to find T directed downward in Lead 1 or in Leads 1 and 2 (Fig 17 D) When right axis deviation is present T is often downward in Leads 2 and 3 (Fig 39 A) In each case the appearance is similar to that seen when one ventricle precedes the other in its contraction because of a lesion of one bundle branch and the T wave lies opposite to the predominant direction of the QRS group in each lead In Chapter III this feature of the T wave of axis deviation curves was discussed in detail and it will suffice here to repeat the conclusion that this change of T is due to something other than the cause of the axis deviation This other factor appears to be an abnormal condition of the ventricular muscle the direction of the axis of QRS apparently determining whether T shall be inverted in Leads 1 and 2 or in Leads 2 and 3 This abnormal condition is sometimes due to the physiological influences of muscle hypertrophy or strain but usually to pathological changes in the tissue

Myocardial changes may lead to inversion of T in all three leads In such records the electrical axis of QRS is usually normal though occasionally right or left axis deviation may be found Abnormal inversion of T may occur in a record in which QRS does not h[illegible] 9 B) This change pro[illegible] process—either toxic or acute inflammatory or chronic sclerotic—which has not involved the bundle branch or Purkinje tissue

Toxic conditions such as uremia and hyperthyroidism cause abnormal inversion of T as may the toxemia of pneumonia or of typhoid fever Certain drugs cause abnormal inversion of T Especially noteworthy because of their common use in cardiac disease are digitalis and quinidine

Mention should be made again here of the interesting small group of individuals without cardiac disease discussed in Chapter II who may show an inverted T and T_3 when in the seated position and a normal T and possibly also T_3 when recumbent

ized shortening of the duration of contraction would also produce a change in the direction of T It seems that a localized area of scarring also may have this effect, though the physiological process involved is not known

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lar T wave to cause a deflection during the early portion of the S T segment has been mentioned on page 37 This must be taken into consideration when examining the S T segment for a deflection due to the auricular T must not be attributed to a peculiarity of the ventricular contraction It is however rare to find this deflection large enough to change the S T junction from a normal to an abnormal position

Certain records show an abnormality of the S T segment which is sometimes the only part of the curve found abnormal In other records the S T segment is affected along with changes in the height of T or in the direction of T or both Abnormality of this portion of the curve was first noticed as a result of the extensive myocardial damage which followed the injection of corrosive substances into the ventricular muscle The changes observed in clinical records are of various kinds

One of the most important abnormal features of the S T segment is an elevation or a depression of the S T junction so that it occurs at a greater than normal distance from the zero level of the record This may sometimes be associated with a T wave on the same side of zero as the displacement of the S T junction as in Figure 42 C and D and sometimes with a T wave on the opposite side as in Figure 42 H

The slightest demonstrable change in the S T segment consists of a filling in of the normal upward or downward concavity which precedes the peak of T so that the line is straight from the S T junction to the peak of T (Fig 42 A and Lead 1 of Fig 75 C) This may be associated with a slight (1 or 2 mm) or more marked displacement of the S T junction in the direction of the peak of T (Fig 42 C) In other cases the S-T segment may be slightly convex upward with an upward T producing a dome shaped wave (Fig 42 B) or downward with a downward T producing an inverted dome These changes in the S T segment also may be associated with a displacement of the S T junction as seen in Figure 42 C and D and in Figures 63 and 65 A more complicated disturbance of the S T segment consists of an initial movement in a direction opposite to that of the peak of T which follows as in Lead 3 of Figure 76 C and Lead EF 3 of Figure 31 A

In precordial leads of normal persons the T wave often is found directed downward in records from the right sternal margin and in normal children and occasionally in normal women from the left sternal margin also Leads from other positions do not show an inverted T wave except when the myocardium is abnormal Rheumatic myocarditis and digitalis may give rise to a downward T wave in precordial leads and the inversion from these causes is likely *to be more marked in records from near the sternum* than from the apex The myocardial degeneration resulting from coronary arteriosclerosis is more frequently the cause of inversion of T in leads from the apex and to the left than in leads from nearer the sternum However T may be inverted by either of these causes in records from all precordial points Other forms of diseases also and toxic conditions may cause inversion of T in one or more precordial leads No relation has been observed be tween the inversion of T in the limb leads and inversion in the precordial leads There may be abnormal inversion in the limb leads and none in the precordial leads or the limb leads may have a normal T and in one or more precordial leads it may be inverted or isoelectric

An isolectric T wave in precordial leads is to be considered as a transition stage between an upward and downward wave and therefore is abnormal Its causes are the same as those of an inverted T

Notching of T is rare in the limb leads This wave is typically a smooth rounded or peaked elevation unless it should have a P wave superimposed upon it as in Figure 45 and Figure 49 A and B Notching of T is seen in Figure 76 C as a result of digi talis and in *Figure 78 B as a result of quinidine* administration A similar notching has been observed in records from certain patients suffering from hyperthyroidism Nothing can be said as to the significance of this notching of T except that it is probably due to a toxic reaction of the myocardium Notch ing of T is also found in records obtained by precordial leads It is much more frequently seen in these leads than in the leads from the extremities Its significance in the precordial leads is not understood but it is thought to be an abnormal finding

Abnormality of the S T segment The tendency of the auricu

which is different in certain features from the deviation of S T found in cases of infarction It is associated with an inversion of T_1 when left axis deviation is present (Fig 35 c) and less commonly with inversion of T_3 either with or without T when right axis deviation is present The lead with downward T often has the S T junction below the zero level and the reciprocal lead 1 or 3 as the case may be has the S T junction above zero At times the junction may lie as much as 3 or 4 mm above or below the zero level In each case the S-T segment describes a normally concave line from its origin at the S T junction to the peak of T differing in this respect from the abnormal S T segment resulting from coronary thrombosis which is often diphasic or is convex in the direction toward the peak of T (Figs 61 A and 62 A)

A deviation of the S T junction similar to that found with hypertrophy is seen in many records with bundle branch block and in these records it is also a permanent feature The presence of a permanent deviation of the S T segment in these two types of records hypertrophy and bundle branch block makes one think that it might be due in each case to a delayed completion of the spreading of the contraction in one ventricle In the hypertrophied heart this would be attributable to the increased muscle mass and in the heart with bundle branch block to the conduction defect

In precordial leads as in the limb leads the S T segment may be affected alone or associated with a T wave abnormality It is affected by much the same things as affect it in the leads from the extremities such as by digitalis quinidine coronary thrombosis acute rheumatic myocarditis and other acute myocardial processes It may be affected differently in the lead from one precordial area than in that from another or it may be similarly affected in leads from all areas depending upon the extent or the character of the myocardial disease

Coronary T wave This term has been applied to an appearance frequently observed in the S T segment and T wave during the healing stage following coronary thrombosis It is important to emphasize that similar features may develop slowly in patients who do not give a history suggesting coronary thrombosis but

This produces a diphasic T wave of one of the general forms shown in Figure 42 F and G or the reverse of these forms so that the first phase is downward and the second upward. If the first

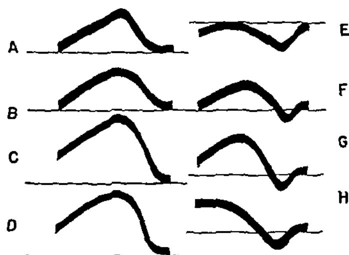


Fig. 42. Abnormalities of the S-T junction, S-T segment and T wave.
A. Straightening of the S-T segment.
B. Convexity of the S-T segment with dome shaped T wave.
C. Elevation of S-T junction with straightening of S-T segment.
D. *Elevation of S-T junction with convexity of S-T segment and dome shaped T wave.*
E. Coronary T wave with upward convexity followed by a downward peak. Note that the upward convexity does not cross the isoelectric level and the wave is therefore not diphasic (+ −).
F. Diphasic T wave (+ −) with the first portion of the S-T segment straight to the first apex of T.
G. Elevation of S-T junction with convexity of the first portion of a diphasic (+ −) T wave.
H. Elevation of S-T junction with an inverted T wave. This T wave is not diphasic because there is no upward peak.

movement does not cross the zero level as in Figure 42 E the wave is not diphasic but still is of the "coronary" type.

Changes in the S-T segment which are due to acute conditions such as pericarditis or cardiac infarction are found to vary from time to time, tending with healing of the disease to return gradually toward a more normal contour. In other hearts when the cause is a more permanent one such as myocardial fibrosis or ventricular hypertrophy, abnormality of the S-T segment may remain for months or years with little change.

Records with a *marked right or left deviation of the axis of QRS* often show a permanent deviation of the S-T junction

which is different in certain features from the deviation of S T found in cases of infarction It is associated with an inversion of T_1 when left axis deviation is present (Fig 85 c) and less commonly with inversion of T_3 either with or without T_2 when right axis deviation is present The lead with downward T often has the S T junction below the zero level and the reciprocal lead 1 or 3 as the case may be has the S T junction above zero At times the junction may lie as much as 3 or 4 mm above or below the zero level In each case the S T segment describes a normally concave line from its origin at the S T junction to the peak of T differing in this respect from the abnormal S T segment resulting from coronary thrombosis which is often diphasic or is convex in the direction toward the peak of T (Figs 64 A and 65 A)

A deviation of the S-T junction similar to that found with hypertrophy is seen in many records with bundle branch block and in these records it is also a permanent feature The presence of a permanent deviation of the S T segment in these two types of records hypertrophy and bundle branch block makes one think that it might be due in each case to a delayed completion of the spreading of the contraction in one ventricle In the hypertrophied heart this would be attributable to the increased muscle mass and in the heart with bundle branch block to the conduction defect

In precordial leads as in the limb leads the S T segment may be affected alone or associated with a T wave abnormality It is affected by much the same things as affect it in the leads from the extremities such as by digitalis quinidine coronary thrombosis acute rheumatic myocarditis and other acute myocardial processes It may be affected differently in the lead from one precordial area than in that from another or it may be similarly affected in leads from all areas depending upon the extent or the character of the myocardial disease

Coronary T wave This term has been applied to an appearance frequently observed in the S T segment and T wave during the healing stage following coronary thrombosis It is important to emphasize that similar features may develop slowly in patients who do not give a history suggesting coronary thrombosis but

who may have had a slowly narrowing coronary branch, sometimes causing the anginal syndrome, sometimes symptoms of most atypical sorts or even without symptoms at all. The coronary T wave may also be found in patients with other forms of acute myocardial disease.

The characteristic feature is that without elevation of the S-T junction *there is an upward convexity of the S-T segment which* carries the record at first slightly above its point of origin and then downward to form an inverted T wave. This is shown in Lead 2 of Figure 64 B and 66 A, Leads 2 and 3 of Figure 66 B. Almost all downward T waves show an upward convexity of this interval, but the feature to be emphasized is that in the coronary T wave the typical upward convexity carries the line of the S-T segment *upward before it goes downward*. The wave may or may not be diphasic (+—), however, depending upon whether the upward movement carries the S-T segment above zero. This important feature of the coronary T wave can be noted by comparing the S-T segment of Lead 2 in Figure 66 A with that in Lead 3. In the former the typical upward convexity is present. In the latter the S-T segment is convex upward but does not show a characteristic upward deflection followed by a downward one. In Lead 3 of this record the S-T segment is at first horizontal and then downward. In Figure 35 B the typical upward deflection appears in all three leads.

The term "cove plane T" has been introduced by Rothschild, Mann and Oppenheimer to describe T waves of this type, indicating a cove followed by a plane. The term adequately describes most of these waves (Figs. 64, 65, 66) but does not apply to others *which are equally diagnostic but have a very short final limb of* T. Moreover, many inverted T waves approximate the appearance of a cove and plane (Lead 1 of Fig. 35 C and Lead 2 of Fig. 39 A) and yet do not fulfill the criteria of the coronary T. Although the term cove plane has the advantage of not suggesting a diagnosis, yet this inapplicability of the term seems to make it undesirable. On the other hand, the coronary T is due to coronary disease in the great majority of cases and affords a satisfactory term to describe the similar T waves which are now known to result from other forms of disease.

The apex of the coronary T wave is turned downward and it may be accompanied by a downward T in the other limb leads though these T waves need not be of the coronary type Its voltage is often considerable and when this is so the resulting peak is sharp in leads which afford an adequate representation of the voltage for example Lead 1 of Figure 68 B The voltage may be unusually large in certain of these cases at times reaching 10 mm Less frequently it may be unusually small If the characteristic features appear only in Lead 3 it cannot be considered to have the significance of the coronary T wave unless T is also turned downward or is isoelectric Such a T wave is occasionally found in Lead 3 of records from normal hearts but it does not have a large amplitude in these records

In precordial leads the coronary T wave may also be observed (Lead CF 5 of Fig 68 A and Leads FF 3 4 F and CF 5 of Fig 68 B) It is especially likely to occur during the healing stage of infarction of the anterior surface of the heart or as a result of a chronic occlusive lesion in this situation It is never found with infarction of the diaphragmatic surface unless the anterior surface is also affected There are other varieties of disease of the myocardium which may give rise to this appearance of the T wave in precordial leads and it may also be caused by drugs In precordial leads its amplitude sometimes reaches great size occasionally as much as 15 mm or more as in Lead 4 F of Figure 68 B

Abnormal U wave In diseased hearts the U wave may be found unaffected or may be inverted Inversion of U has not been reported in records from normal hearts and may occasionally be the sole ele[illegible] tion is [illegible] of T a[illegible] *ssion of a still* further prolongation of this wave This can be determined by comparative measurements of the Q T duration in other leads (Fig 27 B) The U wave has been noted to be infrequently found in records from patients with heart failure In such patients the administration of digitalis is often followed by the appearance of a U wave when it was not present before treatment It has not been observed to be affected by quinidine

In precordial leads the U wave is usually more prominent than in the limb leads It is apt to be particularly large after the administration of digitalis

Summary

Recognizable evidences of the influence of myocardial disease upon the auricular muscle are rare but if either abnormal duration notching *of the peak* or *deep inversion of the P R level* (auricular T) are found disease of the auricular muscle may be diagnosed

When the ventricular muscle is affected by disease or by toxic or abnormal metabolic influences the QRS and T waves are likely to undergo certain changes one or more of which may be present in the same record

1 Notching or slurring of QRS

2 Prolonged duration of QRS

3 A voltage of QRS less than 0 5 millivolts

4 T waves turned downward in Lead 1 or Lead 2 or both

5 A voltage of the T wave of less than 1 millivolt

6 A special peculiarity of the S T interval this portion of the curve originating at an abnormal distance from the zero level and passing to the peak of T by an unusual curve This leads to a deformity of the T wave such as is found especially after coronary thrombosis

7 The S T interval may arise from the proper level but may pass to the peak of T by an abnormal course This also leads to a deformity of the T wave

8 Associated with a downward T wave there may be a special type of deformity of the S T interval called the coronary T wave The S T interval is convex upward passing upward from its point of origin before turning downward to the point of T In the precordial leads

9 An abnormally large Q wave may be present the R wave may fail to appear in leads from positions 3 4 and 5 or may be abnormally small

10 The S T junction may be abnormally elevated above or depressed below the zero level

11 The S T segment and T wave may be abnormal

12 The T wave may be inverted

These features which have been attributed to myocardial disease are occasionally found in records from apparently healthy individuals The frequency of such findings is indicated in the studies by Johnson and by the Metropolitan Life Insurance Company The increased frequency of such findings with increased age is believed to indicate that the cause in the majority of cases is the deficient myocardial circulation which results from coronary arteriosclerosis Other etiological types of myocardial disease may be represented in individual cases but it is probable that such cases are much less frequent than those due to coronary arteriosclerosis To find one or more of these abnormal features in the electrocardiogram should be considered as definite evidence of the presence of myocardial disease The cause of this whether toxic metabolic inflammatory or degenerative must be determined by the other features of the patient s history and examination

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In order to decide that an abnormal electrocardiogram is due to s ructural myocardial disease we must first exclude the pos sibility that the abnormality may be due to a functional disturb ance of the myocardium To do this we must observe the abnor mal feature over a period of time to see if it is constant or if it chan es We must consider the clinical features of the case espe cially in regard to the presence of toxins or evidences of hormonal dysfunction or of vitamin deficiency We must seek for other evi dences of structural cardiac disease such as enlargement or for signs of cardiac insufficiency or the anginal syndrome or the *m urs* of valvular stenosis or insufficiency There are only a few special peculiarities of the electrocardiogram which are defi nitely indicative of myocardial disease Usually the *decision be tween disease or dysfunction* as the cause of an electrocardio graphic abnormality must be made on rather broad lines after a consideration of all other factors in the clinical *picture*

The electrocardiogram *like any other single feature of the clinical* examination is quite insufficient *as an isolated fact to afford a* basis for a prognosis If two hearts have *the same abnor malities in* the record there would still be *a difference* in the *prognosis depending upon the condition of the valves the height of the blood* pressure the character of the life led by the *individual and* so on Yet the electrocardiogram is a very im portant feature for it m y give us the only direct evidence of disease of the ventricular muscle Two patients who were exactly alike *in all respects except their electrocardiographic* records would still be far from *similar if one had* normal ventricular waves while the *other ha* an abnormal record like any of those of Figures 23 , *27 or 31 The* one with *the normal* waves would have *a much better cardiac prognosis* for his ventricular muscle is more likely to be free from *disease*

Notching of the QRS group is *usually* due to a permanent le sion of the *myocardium It is apt* to be a rather *diffuse* myocardial disease and if the *notching of QRS is* persistently associated with small sized waves or with *an abnormally* inverted T *wave we* may feel certain that *the myocardium* is extensively *damaged* Patients showing *this combination of abnormalities* in their rec ords ha e with rare exceptions a *limited* cardiac reserve and do

CHAPTER V

CLINICAL SIGNIFICANCE OF ABNORMAL WAVES

SINCE the electrocardiogram records one of the physiological functions of the heart muscle, it follows that there may be variations in this function which do not result from structural myocardial disease. Local areas of deficient circulation may give rise to functional changes or the whole myocardium may be depressed or stimulated simultaneously. Local areas of functional depression affecting appropriate cardiac areas may give rise to changes in the duration of the P-R interval, changes in the duration and form of the QRS group and of the T wave. Widespread functional depression may decrease the voltage of QRS and T or may cause inversion of the T wave without any basis of structural organic disease. Changes due to functional causes are reversible and disappear when the cause is no longer present. They are thus likely to be of relatively short duration.

Certain forms of acute myocardial disease which may produce abnormalities of the electrocardiogram may be followed by such complete recovery of the tissues that the abnormal features of the electrocardiogram may disappear. Such changes usually take a considerable time. Abnormalities of long duration should be suspected of having an underlying structural organic cause. Even when permanent changes due to structural disease exist there may be superimposed other temporary variations due to variations in the functional condition of the myocardium in the border zone between diseased and healthy tissue. This might occur for example in the periphery of an area of infarction or in an area whose blood supply was so near the limit of physiological necessity that it would be beyond the ability of the coronary flow to meet minor additional demands. The presence of drug effects especially must be carefully excluded when considering the significance of an abnormality of the electrocardiogram.

diac reserve has been seriously taxed Figure 23 A is from such a patient who had no evidence of valvular disease or cardiac en largement These complexes of bundle branch block sometimes appear during pneumonia or other infectious disease The rec ord of Figure 25 B appeared during pneumonia and during the following year changed to the form of Figure 27 A During this year the patient who had neither valvular disease nor cardiac enlargement developed a moderate limitation of his cardiac re serve whereas previous to his illness he had been a very power ful man

The appearance of bundle branch block usually marks a per manent change in the electrocardiogram for the bundle branch usually fails to recover its function and the abnormal complex persists In certain cases bundle branch block is not permanent The curve may change from block to a normal complex and back to block again These changes are due to temporary variations in the function of a diseased bundle branch

Occasionally in patients who developed the condition during an acute illness complete recovery has taken place or the com plex has changed to one showing a less radical disturbance of the mechanism of ventricular contraction for example a notched QRS with an abnormal T or normal QRS with abnormal T This event would indicate a temporary disturbance of the bun dle s function perhaps due to some such process as edema or infil tration with round cells or some other process which could re solve The prognostic importance of such a change in the waves would depend upon the danger of later damage by scar tissue formation In the majority of cases however patients with curves showing bundle branch block are limited in their ability to exercise

Patients with right bundle branch block have been shown by Wood to have a prognosis measured in duration of life and degree of health much better than had previously been con sidered possible Many patients with left bundle branch block have been observed by the author to live for years with compara tive freedom from restriction of their activities and it is believed that a similar careful study of patients with left bundle branch block also would reveal a better than expected prognosis

not improve so as to become even approximately normal Willius has shown that *the average expectation of life is distinctly less* for patients with notching of QRS than for patients otherwise similar but with normal ventricular waves

Notching of QRS may depend upon a relatively slight pathological process affecting a large muscle area In a group of 6 cases whose records showed a typically notched and slurred QRS without abnormality of T Pardee and Price found a diffuse slight myocardial disease in 2 and a moderately severe diffuse disease in 4 Notching of QRS may also be due to disease affecting a primary or a large secondary branch of the A V bundle If such disease should vary in extent or severity from time to time variations in the notching or in the direction of the QRS or T waves might result A variation in the notching may also be due to marked variation in the height of the waves of QRS Large excursions tend to stretch out the notches as it were and to make them less noticeable Small excursions have the reverse effect as may be seen in the records of Figures 24 A and 28 A which are from the same patient The notching signifies the same change in the ventricular muscle whether it is less noticeable in higher waves or more noticeable in smaller ones

Notching may vary from beat to beat in some records in a cycle that coincides with the respiratory movement This will be readily understood to be due to a rotation of the heart by the movements of the diaphragm and is quite independent of cardiac function

Those patients with an electrocardiogram showing *bundle branch block* frequently have a considerably damaged heart The abnormal waves result from a disease which is often extensive and of the chronic sclerotic type Exceptions occur however for a very small lesion if properly placed would affect the function of a bundle branch so as to cause this type of curve *Arteriosclerosis of the artery supplying one bundle* branch may cause a local fibrosis of the branch while the rest of the myocardium might be relatively undamaged

There are many patients who have typical bundle branch block complexes and yet are able to perform an average amount of exercise without signs or symptoms to suggest that their car

If an abnormal inversion of T is accompanied by notching of the QRS group the change is usually a permanent one for it is usually in such cases due to myocardial fibrosis

When no cause for a muscle intoxication is present we must consider an abnormal T to indicate a diffuse myocardial disease The extent of this disease may be inferred by observing whether the voltage of the T wave is of good size and whether the QRS group shows notching or slurring in the course of its waves The inverted T_1 found with marked left axis deviation of QRS is of doubtful significance for it has been found that in 20 per cent of such cases the heart does not show either gross or microscopic myocardial disease The only change is a marked enlargement of the heart so that the abnormality of T must have a functional basis It is believed that the muscle cells suffer from an insufficient blood supply due either to the coronary blood flow being inadequate for the large mass of the hypertrophied ventricles or to the hypertrophied muscle cell being too large for a proper exchange of metabolites between its central portion and the surrounding tissue lymph Each of these factors may contribute to the result in a given case

As to prognosis—a heart with an abnormal T wave is a poorer risk than one without it though if this is the only abnormality revealed by a thorough examination of the heart the patient is not likely to be more than slightly incapacitated Over a period of years such a heart would tend toward gradual failure The follow up reports of the Mayo Clinic show that when associated with other abnormalities of the cardiovascular system T wave abnormality added considerably to the gravity of the prognosis such patients having a shorter average duration of life than those with normal T waves The finding of a downward T and T_3 was less serious in this respect than if T_1 or T_1 and T were downward

The voltage of QRS may vary from time to time with the functional state of the heart muscle but without disease the variations keep within the limits described in Chapter II Variations in the voltage of QRS are only significant if there is no change in the general configuration of the group for a change in the function of intraventricular conduction will change both the height and

The coronary T wave has been found chiefly in patients whose exercise was limited by precordial pain even though the pain might have been so slight as to be described as merely a discomfort Some of them have severe attacks of typical angina pectoris Some die of cardiac infarction A few do not have any trace of pain but have an abnormal amount of dyspnea on exertion With these it is probable that in addition to the focal necrosis resulting from coronary occlusion there is a more extensive myocardial disease The patients are usually able to be about and carry on their work in spite of the pain or the dyspnea Some few of these patients may be entirely free from both pain and dyspnea and may resume their life without symptoms They may live for five years or more after having shown the coronary T wave and during this time the T wave may either remain unchanged or may give place to a normal wave The prognostic significance of the wave returning to normal is not known It has not been determined whether such patients are more or are less likely to have subsequent attacks of coronary thrombosis than if T had remained abnormal They are however, usually found to have improved clinically and are on the average less likely to have the anginal syndrome than one with a persistently abnormal T The reappearance of a normal T would seem to be an evidence of a compensatory increase in the collateral anastomoses of the coronary system so that the affected muscle area has again become properly nourished As a rule however patients who show the coronary T wave have not a normal cardiac reserve and even if they have there is the ever present possibility that a large coronary branch may become occluded with a fatal result

Abnormal inversion of the T wave may result from the action of digitalis and other drugs so that the action of such drugs must be excluded before T wave abnormality can be ascribed to myocardial disease We should obtain another record after the drug has been excreted from the body There are cases with uremia which make it evident that the T wave may be inverted in Lead 1 or Lead 2 by the action of toxins arising in the body These records sometimes show low voltage of the waves as well The inversion disappears and the waves become larger after the cause of the abnormal function is removed be it drug or tissue poison

this is also small in all leads the smallness of T may be due to toxic or metabolic causes or to unusual short circuiting by the tissues around the heart If QRS is normal or large an abnormally small T might indicate a diffuse fibrotic change in the muscle Such a change is not usually severe enough to incapacitate the patient seriously but it is rare to find a small T wave with large QRS excursion from a heart with a normal reserve power This myocardial condition rarely improves though the course need not be rapidly progressive If there is a notching of QRS due to a bundle branch lesion the tendency to a large sized T is so great that even extensive myocardial disease will not produce an abnormally small T unless the heart is in extremis

It seems certain from the many published correlations of the electrocardiogram and the pathological findings in the heart that many varieties of structural myocardial disease will give rise to abnormal changes in the records Certain pathological features are frequently associated with certain features in the curves Cardiac infarction gives rise to a special type of changes in the S T interval A healing infarct or an area of slower degeneration of the muscle gives rise to the coronary T wave Certain less definitely understood pathological changes associated with coronary arteriosclerosis cause the large Q_3 These changes however *must not* be considered pathognomonic of the effects of coronary arteriosclerosis because other types of myocardial disease occasionally give rise to these same electrocardiographic features In *the end it is the* effect upon the muscle fibers in terms of irritation and degeneration—the change in the cellular metabolism—which produces the change in the record It is not difficult therefore to understand how different etiological factors might give rise to a similar state of abnormal cellular metabolism and if in addition to *this the same* anatomical region is affected we can understand the appearance of the same abnormal features in the electrocardiogram

In most cases the abnormal metabolism is due to the presence of pathological tissue in the myocardial structure This may be part of an acute infectious disease like rheumatic fever or an acute degeneration due to *coronary* thrombosis or a slow degeneration due to the more prolonged action of a less acutely de

the form of QRS Notching is apt to be associated with an increased voltage of QRS in the complexes affected by intraventricular conduction defects In the absence of notching abnormally large voltage of QRS may be a sign of a muscle that is hypertrophied

A small voltage of QRS that is between 9 mm and 5 mm may be due to a subnormal functional state of the muscle but the influence of emphysema and of pericardial effusion in decreasing the height of the waves must always be borne in mind If smaller still it is usually so because of a diffuse fibrosis of the muscle Patients with these very small QRS groups are not usually able to carry on normal physical activity and are likely to continue to be thus restricted

When low voltage of QRS is the only abnormal feature of the ventricular complex it is not usually an indication of cardiac disease One small series has been reported by Barritt in which only 35 per cent of the cases were found to have definite evidence of cardiac disease When however low voltage of T was combined with low voltage of QRS 92 per cent had definite evidence of cardiac disease Steuer has reported a series of hospital cases with autopsy 10 per cent of which did not have any cardiac disease and yet had low voltage of QRS It is evident as has been previously stated that low voltage of QRS may have extracardiac causes

The voltage of T has a somewhat different significance from that of the QRS group as is evident from the fact that QRS and T are not always both large or small in the same record A large T may be present because the muscle is normal and is overactive or may be due to hypertrophy of the ventricles Occasionally one sees unusually large T waves in the standard leads after coronary thrombosis or in patients who have not had the symptoms of thrombosis but are suspected of having marked coronary narrowing A large T may also be due to the change in the ventricular contraction produced by a lesion in the bundle branch tissue Coincident signs of this would be noted as notching or increased duration of the QRS group or probably both

In the same way an abnormally small T is of itself an uncertain indication but must be considered in relation to QRS If

A single small lesion which happened to involve one of the bundle branches would cause a marked change in the record while if the same lesion were in the deep layers of the ventricular muscle it probably would not affect the record recognizably Even several foci might fail to affect the electrocardiogram recognizably provided they did not involve important branches of the auriculoventricular system

There have been so few careful autopsy studies of patients with normal electrocardiograms that it is difficult to make a statement as to the amount of generalized ventricular myocardial disease which may exist without affecting the record in one of the recognizable ways Pardee and Price reviewed the literature of such correlations up to January 1938 and reported on the study of 13 cases with a normal ventricular complex 7 of these showed a normal myocardium and the other 6 showed such structural changes as slight increase of fibrous tissue few small areas of fibrosis polymorphonuclear foci with a perivascular distribution and scattered in the interstitial tissue None of these patients was suffering from signs or symptoms of heart disease and except for the one with the polymorphonuclear foci who died of acute bacterial endocarditis all died of non cardiac conditions A review of 47 cases with abnormal complexes combined with 86 other cases collected from the literature leads to the impression that a normal electrocardiogram was not to be obtained from a heart which had more than slight localized or slight diffuse disease of the ventricular muscle an amount which could be determined only by microscopic examination and which would probably not affect the cardiac reserve or cause other symptoms It must be emphasized that a normal electrocardiogram does not indicate the state of the coronary arteries but only of the myocardium Blumgart and his associates and others have demonstrated that it is not infrequent to find a myocardium which is normal or is the seat of only slight patchy fibrosis and yet the coronaries may show one or more occluded branches The collateral circulation in these hearts had become adequate to prevent further degenerative changes in the muscle It is because of this possibility that we should not be too sanguine about the

ficient capillary circulation, as would occur with chronic arterial narrowing A similar altered metabolism may exist in other cases as a pure disturbance of function without there being demonstrable pathological tissue Pardee and Price have reported an instance of this in a patient with coronary arteriosclerosis who showed an abnormal T wave several months before death and then just before death showed a T wave of normal form A careful pathological examination failed to reveal any evidence of myocardial structural disease though coronary arteriosclerosis was present The earlier abnormal T wave must have been due to functional myocardial changes or to a structural change which *had entirely resolved at the time of death The former of these* possibilities seems more likely in view of the demonstration by Blumgart and his associates that the T wave changes resulting from temporary coronary occlusion in animal experiments would disappear when the occlusion was relieved Provided the occlusion had not been maintained for more than a certain time residual *areas of myocardial degeneration were not found when* these hearts were examined after death

In rare chronic coronary cases with congestive failure or the angina of effort structural changes may not be found even after the finding of an abnormal electrocardiogram In these cases the abnormality of the record was probably due to coronary insufficiency causing a subnormal nutrition of the muscle Autopsies on chronic coronary cases with normal myocardium are rare probably because they do not come to autopsy until the abnormal nutritional state due to coronary narrowing has existed long enough to lead to structural changes

The finding of an abnormal electrocardiogram is of great value in helping to decide upon the presence of disease of the ventricular myocardium With the qualifications detailed above we may be sure that when these abnormalities of the record are found the muscle is diseased The different abnormalities of the waves are to be considered not so much as the result of different degrees of myocardial involvement as of a different location or distribution of the process

On the other hand we cannot say that whenever we find *a normal electrocardiogram* we are dealing with a normal muscle

5 The degree of interference with cardiac function
6 The possibility of repair of the structural defects
7 The possibility of restoration of function

Besides these there are many secondary features such as age sex and the demands of life in regard to family and business activity It must be evident that the only thing that can be discussed about the prognostic importance of the electrocardiogram is the part which it may contribute to the prognosis It throws light upon one phase of the prognosis namely the extent of the structural changes in the myocardium Even here there are some defects in the clarity of its light as has been emphasized when considering the abnormalities of the waves that may result from functional myocardial disturbances

On the average patients with bundle branch block complexes and complexes with a low voltage QRS group do not do well These abnormalities result from more severe lesions in the majority of cases but still exceptions occur often enough to make it unsafe for the reputation of the prognosticator to say from the record that this patient will surely be in a serious category Certain patients with inverted T waves in Lead 1 or Lead 2 or both may do very well The majority of them however do not but it is impossible for us to say from the record alone whether the patient is in a good or a bad category The prognosis depends more upon the etiology and the state of the disease than upon the effect it has had upon the electrocardiogram

The coronary T wave tends to return to normal in a considerable number of cases I am not at all certain that the patient who has had this peculiarity and has later lost it is in a better category than another who still has it two years after it is first discovered There is danger from coronary thrombosis in each case for we know that the patient s coronary arteries are sclerotic Some patients with the coronary T wave may have pain on effort some may be free from pain but I do not believe that in either case they have more than a slightly greater likelihood of a severe attack of thrombosis than other patients with similar symptoms whose T wave might be normal

The electrocardiogram is a physical sign telling certain things

condition of a heart suspected of coronary arteriosclerosis merely on the basis of a normal electrocardiogram

It must be recalled that *although a record may be a normal* one yet it may not be normal for the individual in question Certain patients who have had a cardiac infarction and shown the *electrocardiographic changes* characteristic of this may after a period of a year or two give an electrocardiogram which taken by itself must be *considered* normal Comparison with a record taken before the attack will often show a difference in either QRS or *T* or perhaps in both which will indicate that the record in question is not normal for the particular individual

Prognosis

Even at the expense of some repetition it seems worth while to emphasize here the prognostic aspect of an electrocardiographic record It is unfortunate that the physician is so often *called upon to read the future but it is even more unfortunate* that he so often believes that he can do this by the observation of some one striking feature of the examination Prognosis is not *a matter of exact prediction but of probability and should* always be carefully explained to the patient on this basis A hundred patients of a certain age with a certain type of disease may show an average duration of life which is definitely *less than* the normal for their age but to apply these averages to a particular patient is mathematically unsound and it seems to me morally wrong

A patient may have a much shorter life than the average found with his condition or he may live much longer His limitation may be much more marked than is usual for his disease or he may be practically without limitation of his activity Only after *weeks or months of observation and treatment* can we even approximate the outlook for a particular case

A true prognosis must consider at least a number of factors

1 The etiology of the disease
2 What is its rate of progress (activity)?
3 Can it be checked by treatment?
4 The present extent of the disease

CHAPTER VI

DISTURBANCES IN RATE OR RHYTHM

OUR knowledge of the disturbances in the rate and rhythm of the heartbeat begun by the polygraph has been greatly advanced by the electrocardiograph because this instrument not only gives the time relations of the contractions of the auricles and the ventricles but indicates by the form of the waves when the contraction passes normally over these chambers and when it does not

SINUS DEPRESSION

Sinus arrhythmia The simplest disturbance of the cardiac mechanism is an irregularity which arises from more or less rhythmic variations in the activity of the vagus This is called sinus arrhythmia because the vagus acts upon the sino auricular node (the sinus node) which originates the normal heartbeat retarding or accelerating its activity according as the vagus is more or is less active The contraction stimulus starts at its normal place in the sinus node and passes normally throughout auricles A V node and bundle to the ventricles The auricles and ventricles therefore contract normally and the form of their waves is normal Their rhythm however is irregular as seen in Figure 36 A because the sinus node originates its impulses irregularly

The most common type of sinus arrhythmia is due to an increase of vagus activity with slowing of the heartbeat during the expiratory phase of respiration and a decrease of vagus activity with acceleration of the heart during inspiration as seen in Figure 43 A A slight degree of this sort of irregularity is very common

Almost any record will show variations of 0 02 to 0 04 second in the intersystolic interval but even more marked variations are by no means uncommon especially with the heart rate under

about the myocardium just as the stethoscope tells certain things about the valves In each case it is better to have a normal than an abnormal finding and in each case there may occasionally be normal findings when we believe for other reasons that disease is present Certain patients with valvular disease live to a good *old age and it is possible we shall find* as our experience increases that certain patients with myocardial disease do likewise In each case the positive finding is only one feature of the patient s prognosis and must be evaluated as such

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Fig. 19. Vagal arrhythmia.

A. Record of a rec... of ... ing ... arrhythmia. The P wave ... always precedes the QRS group by the ... interval but the interval between ... cons... ly varies. Note the slightly different form of the fifth, sixth, fourteenth and fifteenth P waves. The ... of ... lengthening ... than the ... The phase ... of respiration ... marked at the top of the record and it can be seen that the ... follows ... Note also the variation in the height of the R waves with the respiratory phase, these being larger at full inspiration and smaller at expiration.

B. ... smaller record of sinus arrhythmia, the phases of slowing and acceleration being quite independent of respiration.

C. Sino-auricular block. The long pauses between auricular waves will be found to be exactly twice the interval between the P waves at the faster rate. The seventh ventricular complex is an instance of ventricular escape. The preceding pause is so long that the ventricular irritability ... and ... comes ... before its time, beginning just after the peak of the delayed P wave.

D. An instance of sinus bradycardia (37 per minute). There is also a sinus arrhythmia.

E. An instance of sinus slowing followed by ventricular escape. The seventh, eighth and ninth ventricular contractions begin before the respective P waves, but the tenth follows after a normal interval. (The tenth cycle is incorrectly marked, having the P wave fused with T instead of with R as ... indicated.)

80 especially also in children, and in people over fifty years of age In Figure 43 A the rate variations can be seen to coincide with the variations in the QRS group due to the movements of the diaphragm In another form of sinus arrhythmia the variations in vagus activity extend over longer periods than the respiratory cycle (Fig 43 B) perhaps lasting even ten or twenty seconds to each phase In sinus arrhythmia the ventricular irregularity is only secondary for each beat follows the auricular beat in the usual way after a normal interval It is the auricular beats themselves that are primarily irregular

A characteristic feature of the electrocardiogram with sinus arrhythmia is that the waves *do not change along with the arrhythmia* The ventricular waves are *usually* of normal form or may show any sort of abnormality Whatever they may be they *do not vary throughout the record* This is also usually true of the P waves for the sinus node continues to originate auricular contractions in the normal way and the spread of the contraction wave is unaffected by the vagus activity In this feature sinus arrhythmia differs from premature contractions of either auricular or ventricular origin for these are associated with an abnormal site of origin of the contraction in the chamber which is premature and with an abnormal form of the electrical curve of this chamber

Certain rare records showing sinus arrhythmia however will be seen to have a slight variation in the form of P coincident with the variations in rate This is more usual in the type with long phases of slowing and acceleration than in that which follows the cycle of respiration It is seen in four of the P waves of Figure 43 A and is due to the vagus activity displacing the site of impulse formation from the more irritable upper portions of the sinus node to the less irritable lower portions As the node is from 3 to 5 cm in length this would cause the impulse to enter the auricle in a slightly different place and would thus change slightly the path of the contraction wave This is the phenomenon of *shifting* or *wandering pacemaker*

Occasionally the site of the pacemaker may be shifted so far downward as to reach the auriculoventricular node There will then be a gradual change of the P wave to an inverted form in

that it seems as though some local disease condition had lowered the irritability of the node. Perhaps a deficient blood supply to the sinus node due to arterial narrowing might be responsible.

Figure 43 F shows another occasional result of excessive sinus depression. The auricular activity is so very slow, due to a marked sinus arrhythmia during the latter part of the record, that the ventricles beat without an impulse from the auricles, being unable to tolerate such a long rest. This phenomenon of *ventricular escape*, shown in diagrammatic form in Figure 46 D, is a very interesting demonstration of the heart's inherent tendency to beat. The ventricular complexes have throughout the form which is normal for the individual of this record, indicating that their contraction stimulus is received along the normal paths. This is usual when the auricular stimulus fails, for the rhythm producing function is taken over by the auriculoventricular junctional tissues, probably by the A V node. The contraction impulse is thus distributed normally over the ventricles and produces the normal ventricular complex of the individual. Figure 45 C also shows ventricular escape in the seventh ventricular complex. Often the ventricular complexes showing the phenomenon of escape are slightly different from those resulting from an impulse from the auricles. This slightly aberrant feature (page 188) is probably due to the fact that the abnormal stimulus does not pass into the branches of the A V bundle quite as does the

[illegible] An au[illegible] ulo[v]entricular nodal rhythm is interrupted [illegible] the [illegible] of the record by [illegible]

Leads 2 and 3 with a coincident shortening of the A V conduction time Eventually when the impulse comes from the A V node the condition becomes an A V nodal rhythm (page 181)

Sino auricular block Is another curious and rather uncommon variety of arrhythmia It is evidenced by the appearance of a normal series of auricular and ventricular waves from which occasionally one complete *heart cycle will be missed the next* one following after an interval almost exactly double the normal interval (Figs 16 B and 43 C) Such doubling of the interval occasionally *persists for a time leading to a halved heart rate* The mechanism of this dropping of a heart cycle is supposed to be a physiological blocking of the impulse which normally passes *from the sinus node to the auricle* so that this *fails to receive* a stimulus from the node and accordingly does not beat until the next stimulus arrives It seems that this mechanism at times may *be due to an inhibition of the physiological stimulus for the* pause is not always as much as twice the normal interval In certain records however the interval between beats is exactly double the usual rhythm interval and instances of a pause exactly three or four times the rhythm interval may be found

Sinus bradycardia Some individuals have a persistently slow heart rate less than 60 per minute Occasionally the heart rate is considerably less than this when at rest perhaps 55 or 50 or rarely 40 per minute and does not increase with exercise to the same extent as in those with a more normal rate when resting The electrocardiogram with this slow rhythm is quite normal in every way except in the matter of the heart rate Figure 43 D is a record from such a patient the rate being 37 per minute— as slow as found with heart block As the P waves and the ventricular waves are quite normal in all three leads showing a normal path of the contraction wave and therefore a normal heart mechanism it is proved that the slowed heart rate is merely due to a slowed stimulus production in the sinus node This slow heart action like sinus irregularity arises from overactivity of the vagus and we often see in such records a slight variation in the diastolic pauses so that sinus arrhythmia is also present This is the case in the figure In certain older patients especially in those past fifty years the slow rate may be so very persistent

give rise to an irregularity of the ventricular systole and may produce only a very small pulse or none at all depending upon how much blood has passed into the left ventricle since the preceding systole that is upon the degree of the prematurity A premature beat may originate in any part of the auricles or the ventricles or in the auriculoventricular system which connects these parts of the heart It is conceivable that the sinus node could originate premature beats but such an occurrence has not come to my attention In any case the electrocardiogram will always indicate the part of the heart from which the premature beats arise

Premature beats originating in the *sinus node* would be difficult to diagnose from a sinus arrhythmia I am not sure that it would be possible to differentiate between them except that the premature beats would occur occasionally during an otherwise regular rhythm while sinus arrhythmia would tend to show a continual phasic variation in the length of successive diastolic pauses If a record is found which shows a premature P wave of normal form in all three leads then this premature auricular beat must be considered to arise in the *sinus node*

Auricular premature beats The diagnostic feature of this condition is the premature occurrence of a P wave of different form from the others of the record in question It is due to a contraction originating at an abnormal focus within the auricles at a time previous to the entry of the normal stimulus from the sinus node (Fig 46 F) The abnormal focus is called *ectopic* because it lies outside of the sino-auricular node where the normal impulses originate This *ectopic* contraction starting at an abnormal point spreads over the auricles by an abnormal path and produces an abnormal P wave The abnormal form of P enables us to recognize a beat as ectopic even if its prematurity is only a matter of a few hundredths of a second

Often as in Figure 5, the first manifestation of abnormality of the curve may be a distortion of the T wave of an otherwise normal ventricular group by the premature P wave falling with T This occurs in all three leads of this record If the auricular beat is not so early it will be plainly seen in the diastolic interval as in Lead 2 of this record The second premature beat in this lead can be observed to have a P wave of different form

normal stimulus and thus is not spread throughout the ventricles in quite the normal manner

Rarely there will be periods of considerable duration when the S A node is depressed and the junctional tissues take over the rhythm producing function at a much slower than normal rate When *'A V nodal rhythm,* shown diagrammatically in Figure 16 c is in effect the auricles may contract in response to a stimulus which arises in the sino auricular node at the same time that a ventricular stimulus arises in the A V node This is seen in the sixth and seventh heart cycles of Figure 44 and results in *the R waves and P waves being superimposed the P waves having* their normal form In other cases the auricles contract in response to a stimulus which passes back from the A V node the P wave then falls just after the QRS group as in the first second third and last complexes of Figure 44 and has an abnormal form usually inverted in Leads 2 and 3 because of the abnormal path *by which the stimulus enters the auricles The ventricular com*plexes of the nodal rhythm are apt to show slight variations of QRS and of T of the type described as aberrant complexes (page 188)

Auricular standstill Auricular standstill is a disturbance of cardiac rhythm in which the auricles cease to beat the electrocardiographic *tracing failing to show evidence of auricular ac*tivity in any lead This arrhythmia is probably not a disturbance of vagal function The ventricles beat with an independent regular rhythm usually slow and the ventricular complexes usually originating in the A V node have the appearance of supraventricular origin Occasionally isolated P waves followed *by ventricular complexes may occur It is usually* although not always a drug effect and either digitalis or quinidine may be the offender It has been observed however in acute cardiac inflammatory disease without either of these drugs having been administered

Premature Beats

Another important disturbance of the cardiac mechanism is due to premature beats sometimes called extrasystoles These

lowed by a prolonged conduction time or by a complete failure of ventricular systole due to a complete blocking of the impulse

There is one circumstance under which a premature auricular beat may fail to be followed by a ventricular beat without reflecting on the integrity of the auriculoventricular system When the auricular beat is so premature that the stimulus it sends to the ventricles arrives there before they have relaxed from the preceding systole they will fail to respond to it The ventricles are refractory to stimuli while contracting so that this impulse from the auricles fails to cause a ventricular contraction even though it has passed normally along the bundle This is why the P waves during ventricular escape in Figure 39 E are not followed by another ventricular beat The auricular stimuli arrives when the ventricles are still contracting

The ventricular complex following the premature auricular beat always has approximately the same form as the other ventricular complexes of this individual Lewis introduced the term supraventricular impulse to apply to one that originated above the branching of the auriculoventricular bundle The ventricular complex resulting from such an impulse called a supraventricular complex is characterized by the usual duration and appearance of the QRS group and the usual form of T found in the individual record in question Auricular premature beats are followed by a ventricular complex of the supraventricular form but it is always slightly different from the other ventricular complexes of the record as can be seen in Figure 43 This difference is evident in records showing the auricular premature beats in three leads The curve may be but little changed in any one lead and yet *quite* a variant in the other two Both QRS and T are changed though as a rule QRS is more evidently so The change in the QRS group depends upon an abnormal spreading of the contraction in the ventricles and this causes a change in the contraction itself which to a greater or lesser extent affects the T wave

Lewis has suggested that the change in QRS is due to a slight delay in the conduction along either the right or the left branch of the AV bundle or to a delay in conduction along some of the finer arborizations of the bundle branches upon the inner

from the normal P waves of this record and yet to follow sooner after the preceding T wave than do the normal P waves If the P waves of the other premature auricular contractions of this

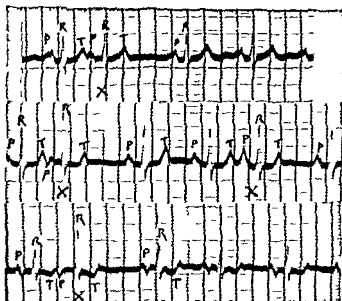


Fig 51 Three leads of a record showing auricular premature beats The premature P waves are at the points marked x and have a different form from those of the other auricular contractions There are two premature contractions in Lead 2 each from a different auricular focus as shown by the different forms of P The ventricular complexes of each premature beat are aberrant

record could be separated from the T wave and considered individually, they too would be found to be different in form from the normal P waves of this case The normal P of this record is upward broad and notched in Leads 1 and 2 while the premature P which is superimposed on the T is not so high or broad in Lead 1 and is downward in Lead 2 Note also the abnormal forms of the various premature P waves of Figure 49 A and B

In each instance in the illustrations the ventricular complex follows the premature auricular beat after a normal P R interval The conduction time after these premature auricular beats is a severe test of the ability of the A V conduction system for the rest period since it last functioned is much shorter than usual A latent defect in the conduction system will sometimes be made evident only by the fact that a premature auricular beat is fol

auricular contraction is nearly always less than that of two normal intervals in the same record

The post-extrasystolic pause is usually greater than the normal interval between beats The increase in the duration has been explained as due to the remoteness from the sinus node of the focus starting the premature beat The longer the post extrasystolic pause the more remote the focus

Ventricular premature beats When premature beats arise in the ventricles they can be recognized by the appearance of waves which have certain distinctive features shown in Figures 47 and 48 There is first a wide QRS group usually with large excursions in the favorable leads and usually with notching or slurring on one or both of its sides or at its peak The duration of QRS is between 0 14 and 0 20 second It is followed by a T wave usually of large size in the favorable leads and usually directed opposite to the chief QRS excursion except in leads in which the initial deflections are unfavorably recorded showing a small height in proportion to the height of the waves of QRS in the other leads This is the case in Lead 2 of Figure 47 B In the unfavorable lead the T wave may be diphasic or absent or in the same direction as the largest wave of QRS in this lead or in the opposite direction In unfavorable leads the QRS group very often is notched or has both upward and downward waves

These complexes are not usually preceded by any indication of a P wave or if they are as in record A of Figure 48 it is at so short an interval that it is plain that A V conduction had not taken place before the onset of the ventricular contraction

An auricular wave occurs at the proper rhythmic interval after the preceding P wave and may fall with the QRS group of the premature beat or with the T wave of this complex It can often be seen as a slight notching or deformity of the curve of the premature beat This submerged P though sometimes quite plain is sometimes scarcely visible especially if it falls during the QRS group (Fig 47 A) The next P wave following the premature beat comes at the proper interval from the submerged P and is followed by a normal ventricular complex the rhythm of the auricles is unaffected The pause after the premature beat the post extrasystolic pause is *compensatory* in the sense that

wall of either ventricle. He has called these contractions arising in this way, *aberrant contractions,* and the ventricular complexes to which they give rise *aberrant complexes.* This aberration he believes occurs after premature auricular beats because their prematurity does not allow the conducting tissues the usual rest period before they are again called upon to function. Such an explanation would point to a slight abnormality of the A-V system at some localized area or at least to an abnormal physiology in one branch of the bundle. It is true that, according to our theories of the electrocardiogram, such a delay in conduction would cause the type of changes in the ventricular complexes which we find in these *aberrant contractions.* From another viewpoint, however, *it is difficult to accept this explanation for it* demands that we believe that *all* patients who have premature auricular contractions have an abnormality either of function or of structural disease in the A-V conduction system. It is curious also that these aberrant complexes are so rare when heart block is present with dropped beats, a condition which we know to be associated with disease in the A-V system. Moreover, aberrant complexes are common in normal dogs with artificially induced premature auricular contractions, and it is difficult to find a reason why there should be a localized abnormality of the A-V system in normal dogs even when under an anesthetic.

A better explanation of this aberrant feature I believe may be found in the abnormal character of the premature auricular beat itself. The contraction does not involve the auricles in the normal way and so may affect the A-V node abnormally. As a result of this the impulse may fail to leave it normally and so to pass down the two branches of the bundle simultaneously as it should. The resulting asynchronous stimulation of the two ventricles would cause the change in the ventricular complex.

The pause after a premature auricular beat, before the next auricular systole supervenes, is always at least slightly longer than the normal intersystolic interval for the record, but is seldom long enough entirely to *compensate* for the prematurity of the beat which started the disturbance (Fig. 46 F). The space of the two intervals from the last normal auricular contraction to the premature contraction and from this to the next normal

auricular contraction is nearly always less than that of two normal intervals in the same record

The post-extrasystolic pause is usually greater than the normal interval between beats The increase in the duration has been explained as due to the remoteness from the sinus node of the focus starting the premature beat The longer the post extrasystolic pause the more remote the focus

Ventricular premature beats When premature beats arise in the ventricles they can be recognized by the appearance of waves which have certain distinctive features shown in Figures 47 and 48 There is first a wide QRS group usually with large excursions in the favorable leads and usually with notching or slurring on one or both of its sides or at its peak The duration of QRS is between 0 14 and 0 20 second It is followed by a T wave usually of large size in the favorable leads and usually directed opposite to the chief QRS excursion except in leads in which the initial deflections are unfavorably recorded showing a small height in proportion to the height of the waves of QRS in the other leads This is the case in Lead 2 of Figure 47 B In the unfavorable lead the T wave may be diphasic or absent or in the same direction as the largest wave of QRS in this lead or in the opposite direction In unfavorable leads the QRS group very often is notched or has both upward and downward waves

These complexes are not usually preceded by any indication of a P wave or if they are as in record A of Figure 48 it is at so short an interval that it is plain that A V conduction had not taken place before the onset of the ventricular contraction

An auricular wave occurs at the proper rhythmic interval after the preceding P wave and may fall with the QRS group of the premature beat or with the T wave of this complex It can often be seen as a slight notching or deformity of the curve of the premature beat This submerged P though sometimes quite plain is sometimes scarcely visible especially if it falls during the QRS group (Fig 47 A) The next P wave following the premature beat comes at the proper interval from the submerged P and is followed by a normal ventricular complex the rhythm of the auricles is unaffected The pause after the premature beat the post extrasystolic pause is *compensatory* in the sense that

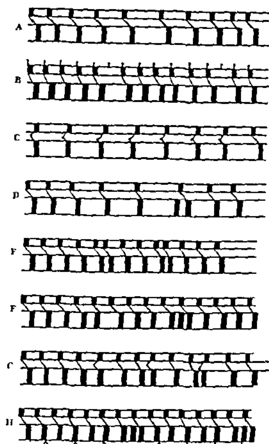


Fig 46 Diagrams illustrating the mechanism of certain arrhythmias The upper block represents the auricular contraction the connecting line auriculoventricular conduction and the lower block the ventricular contraction

A SINUS ARRHYTHMIA The auricles show a phasic variation in the diastolic interval The A V conduction is normal so that the ventricles follow the auricular sequence

B SINO AURICULAR BLOCK The small dot above the auricular rectangle indicates the sinus and the line between the dot and the auricular rectangle indicates sino auricular transmission The auricles beat almost regularly with a slight sinus arrhythmia At two points the sino auricular impulse is blocked so that a complete auriculoventricular sequence is missing

C NODAL RHYTHM The impulse originates between the auricles and ventricles in the A V node It is conducted in both directions so that in the first two beats the auricles and ventricles contract simultaneously In the third and fourth beats the stimulus enters the ventricles before entering the auricles and the ventricles contract first The next three beats show coincident auricular and ventricular contractions and the last beat shows a resumption of a normal sinus rhythm with normal A V conduction

D SINUS ARRHYTHMIA combined with VENTRICULAR ESCAPE The sinus arrhythmia is shown by the phasic variation in the diastolic pause between auricular beats The fifth ventricular beat begins before the stimulus from the auricles has reached the ventricles and is therefore an instance of ventricular escape The next interauricular pause is so long that the mechanism of ventricular escape produces a beat which is not preceded by an auricular contraction and which is followed by the auricular beat of the sinus rhythm This in its turn produces

it is sufficiently longer than the usual diastolic pause of the record to *compensate exactly* for the prematurity of the beat (Fig 46 F) The time between this post-extrasystolic beat and the preceding *normal* ventricular contraction exactly equals two normal intervals In records c of Figures 47 and 48 there are no P waves because auricular fibrillation is present and there cannot be a compensatory pause as there is no regular intersystolic interval Even with auricular fibrillation though the next systole after a premature beat is usually preceded by a diastole longer than the one before the premature beat

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another ventricular contraction soon after that which was produced by the escape mechanism

F AURICULAR PREMATURE BEATS The sixth auricular beat is premature and is followed after a normal conduction by a ventricular beat The post-extrasystolic pause is slightly longer than the normal interval between beats The same process occurs after the ninth auricular beat

F VENTRICULAR PREMATURE BEATS The auriculoventricular sequence is normal except for the fifth ventricular beat which arises prematurely and is *almost completed* before the stimulus from the auricles arrives The next auricular stimulus produces a beat in the normal manner so that the post extrasystolic pause is compensatory The tenth ventricular beat is premature and is *completed* before the normal auricular impulse has arrived The normal A V sequence is not interrupted so that this is an interpolated *ventricular premature beat*

C NODAL PREMATURE BEATS. The fifth auricular beat *originates in* the normal manner *The fifth* ventricular beat originates in response to an impulse *from the A V node* This impulse *is conducted* backward toward the auricles but does not affect them because they are already contracting in response to their own stimulus which has been liberated at the *normal time*

The eighth ventricular beat is *initiated* by an impulse from the A V node which travelling backward stimulates the *auricles before* their normal stimulus has arrived and at the *same time* as the ventricles are stimulated Because the auricular rhythm *is interrupted* the following pause is not fully compensatory

The eleventh ventricular beat is the result of an *impulse* which starts in the A V node and reaches the ventricles earlier than it reaches the auricles It reaches the auricles however earlier than does the *normal auricular* stimulus so that the auricles are activated by the *retrograde* conduction Because the *auricular* beat is interrupted *the following* pause is *not fully* compensatory

F PARASYSTOLE Showing how a parasystolic rhythm may produce ventricular premature beats provided that it is assumed that the *normal* beats do not interfere with the building up of the parasystolic rhythm The arrows below the line of the ventricular beats *indicate* the time at which the parasystolic rhythm becomes effective The first two impulses find the ventricles refractory the third impulse produces a slightly premature ventricular *beat* the fourth impulse produces an interpolated ventricular beat the fifth and sixth impulses again find the ventricles refractory the seventh produces a slightly early ventricular beat and the ninth again an interpolated beat This makes it apparent wh[illegible] rhythms are likely to produce regularly [illegible]

[illegible] degree of sinus arrhythmia [illegible] a slight arrhythmia the regular occ[illegible] slightly

In a patient with a slow heart rate a premature ventricular beat may occur and be completed before the next contraction *stimulus arrives from the auricles* thus the premature beat is

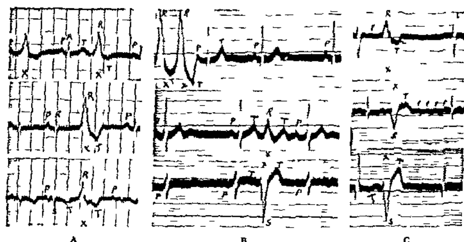


Fig 47 Records of ventricular premature beats (marked *x*) arising in the right ventricle In record A the site of the origin must be high up on the right of the conus arteriosus In record C it must be low down and toward the base of the right ventricle In record B it must be intermediate between these positions

The other ventricular complexes of record B indicate a slight left axis deviation of QRS those of record C a definite right axis deviation Record C also indicates that auricular fibrillation is present

truly an *extrasystole* being *interpolated* between two normal beats (Fig 46 F) This occurs in record B of Figure 47 which record also shows a sinus arrhythmia Such *interpolated* beats rarely if ever result in a pulse wave as they are too premature The ventricular filling is so incomplete early in diastole that the blood ejected by the systole does not cause a pulse of appreciable size

The ventricular complex of premature beats arising in the ventricles has an abnormal form for a reason similar to that causing the P waves of auricular premature beats to have an *abnormal form* *The contraction wave arises in the ventricles* at an abnormal point It is ectopic and therefore spreads over the ventricles in an abnormal manner It is because of this abnormal form that we are able to recognize these premature beats in the presence of auricular fibrillation (Figs 47 C and 48 C) a rather difficult distinction in polygraph records

The increased duration of the QRS of these beats is produced

by a similar mechanism to that causing an increased duration of the QRS of bundle branch block. It is due to a delay in the spreading of the contraction over the ventricles which in turn



Fig 48 Records of ventricular premature beats (marked x) arising in the left ventricle. That in record A must arise high up and near the basal region. That in record C must arise low down near the apex of the left ventricle, while that of record B is from an intermediate position.

is due to its spreading from one ventricle to the other through the muscle of the septum. If the stimulus of the premature beat starts in the muscle it probably passes first to the Purkinje tissue of the ventricle, involves that ventricle in the contraction, and then having spread through the septal muscle to the Purkinje tissue of the other ventricle extends through this to the ventricular muscle on this side.

The QRS groups of these premature beats vary in their relative size and in their direction in the three leads. It is probably possible to determine from the direction of the waves in the three leads the approximate region of the heart in which the beat originates. Our knowledge of the localization of ventricular premature beats in the human heart is based upon records which have been obtained by electrically stimulating the ventricles at various points so as to produce premature beats. This was done originally for the human heart by Barker, Macleod and

Alexander and has been repeated by several other observers There are slight discrepancies in the results obtained by the different observers but these occur only in the results of stimulating the inaccessible posterior portions of the left ventricle and it is possible that the location ascribed to the stimulating electrode may not have been exactly correct

In the case reported by Prinzmetal and Oppenheimer the patient was lying in a right lateral position and the heart was approached by the left transpleural route so that the evident deformity of the complexes of the normal beats which resulted from this change in the position of the heart probably is accompanied by a change in the form of the abnormal beats also

Certain observers have attempted to stimulate the heart mechanically using the percussion hammer through the chest wall after the costal cartilages and part of the sternum and ribs have been removed surgically Such experiments have often shown curves at variance with the results of direct electrical stimulation of the heart It does not seem that the localization of such mechanical stimuli can be exact enough to be used as a basis for far reaching conclusions not only must there be some doubt as to the exact portion of the heart beneath the percussion hammer but it is even possible that some other portion of the heart may respond to the shock of the mechanical stimulus and initiate the extrasystole at a point remote from that of contact

Kountz and his associates have stimulated the revived human heart lying within the thorax and their findings were in agreement with the findings of Barker and his associates They found the initial ventricular deflection upright in all three leads after stimulation of the conus of the right ventricle By stimulation of other points of the right ventricle they produced curves in which the initial deflection was upward in Lead I and downward in Lead 3 Curves with the initial deflection downward in all three leads were obtained after stimulation in the region of the apex of the left ventricle but other portions of the left ventricle gave rise to curves in which the initial deflection was downward in Lead I and upward in Lead 3

Some excellent work has been performed on animals attempting to learn the character of the curve to be expected of prema

ture beats from certain portions of the heart, and these findings too are in general agreement with the results obtained after stimulation of the human heart provided that due consideration is given to the different configuration of the heart in man and in the experimental animal and to its different position within the thorax.

Stimulation of points on the surface of the right ventricle of the living human heart has uniformly produced a QRS group which was directed upward in Lead 1 and, except for the region of the pulmonary conus, was directed downward in Lead 3. On stimulating this latter region QRS 3 was also directed upward. Stimulation of points on the surface of the left ventricle has given rise to a QRS group directed downward in Lead 1 (except for one observation which gave rise to an upward QRS in this lead) and usually upward in Lead 3, although stimulation of the extreme apex of the left ventricle has given a chiefly downward QRS deflection in this lead. In line with the animal experiments and as originally suggested by Einthoven, it seems possible to draw a dividing line across the heart for each lead so that points of stimulation lying on one side of this line will give rise to an upward QRS deflection while points of stimulation on the opposite side will give rise to a downward deflection. Einthoven suggested that such a beat as shown by Figure 47 C would originate at a point more toward the right than the left half of the heart, more toward the apex than the base, and more toward the lower than the upper half of the heart. Such a point would lie to the right of the apex of the ventricles ("rechts bei der Herzspitze"). On the *anterior surface of the heart* the dividing line for Lead 1 seems to run approximately along the right side of the interventricular septum, points to the right of this giving an upward deflection, points to the left a downward deflection. The dividing line for Lead 2 seems to run diagonally across the heart from the lowest part of the right auriculoventricular junction to the middle of the left border, points above this line giving a chiefly upward deflection of the QRS group, points below this a chiefly downward deflection. The line for Lead 3 seems to cross the heart from the middle of the left border to the upper part of the right auriculoventricular junction

stimulation of points above this line giving a chiefly upward de flection of QRS points below this a chiefly downward deflec tion It is probable that similar lines exist *on the posterior surface* of the heart but they cannot be indicated at present

Wilson s theoretical consideration of the relation of the direc tion of QRS and of T to the average direction of propagation of the contraction stimulus (page 320) suggests that the lines of *division between upward and downward deflections on the pos* terior surface of the heart might approximate the projection upon the posterior surface of the lines of division found for the anterior surface The relation of the two ventricles to the pos terior surface is very different from their relation to the anterior surface there being very little right ventricle present and this only at the extreme right inferior portion Because of this it would seem that a premature beat like that of Figure 47 B might arise in the caudal portion of the right ventricle anteriorly or in a right caudal portion of the posterior wall of the left ventricle posteriorly It may be that for this reason it is not possible to describe the site of origin of ventricular premature beats except in relation to the ventricular mass as a whole and particularly not to indicate the ventricle of origin This matter however needs further investigation and must be left undecided on the basis of our present knowledge

It is quite evident from various experiments that any move ment of the heart which displaces it from its normal position *will have an effect upon the form of the waves of the normal* electrocardiogram and therefore upon the waves of ventricular premature beats It is probable that ventricular hypertrophy may have an effect upon the form of ventricular premature beats though this effect cannot be definitely stated

Figure 47 shows *3 types of ventricular premature* beats com monly considered to have an origin in the right ventricle The QRS group of these beats shows a normal or left axis deviation with the chief deflection upward in Lead 1 In Leads 2 and 3 the chief deflection may be either upward or downward de pending upon whether the contraction starts more toward the base and the upper portion of the heart or more toward the apex and diaphragmatic portion

The T waves of these curves are directed opposite to the chief deflection of QRS except in unfavorable leads which give only a small deflection of QRS. In such leads T will be either very small, possibly diphasic or may be in the same or the opposite direction to the chief deflection of QRS.

The type of curve generally believed to be produced by a premature beat starting in the left ventricle is shown in Figure 48. The major deflections of the QRS group are downward in Lead 1. In Leads 2 and 3, as with beats starting in the right ventricle, the major deflections may be either upward or downward depending upon whether the contraction starts more toward the base and upper portion of the heart or more toward the apex and diaphragmatic portion. It must be remembered that the localization of left ventricular premature beats is not as well proven as that of beats starting in the right ventricle because the location of the points of experimental stimulation of the left ventricle affords great technical difficulty since they lie mostly on the posterior surface of the heart. The T wave of left ventricular premature beats is upward in Lead 1 and in the other two leads depends for its direction upon the direction of QRS as has been described for premature beats starting on the right side.

This localization of the site of origin of ventricular premature beats is in accord with the evidence which has been mentioned when discussing the localization of bundle branch block.

The previous beliefs as to the localization of premature beats were dependent upon a misinterpretation of the results obtained by stimulating the ventricles of the dog. There are great differences between the human and the dog's heart in the anatomical relations of the ventricles to each other and in the relation of the heart to the chest wall and to the longitudinal axis of the body. The form of the

portions of the d

the result of sti corresponding points of the human heart that they do not afford a proper guide to the localization of premature beats in man. It is of interest, nevertheless, that the experiments of Rothberger and Winterberg when interpreted with due consideration to the relation of Leads 1, 2 and 3 to the heart

of the dog give results quite in harmony with the present localization of ventricular premature beats

Ectopic ventricular beats are believed to be initiated usually in the Purkinje fibers though probably never in fibers which are far from the junction with the ventricular muscle Less often they may arise in the ventricular muscle itself The curves of such beats as are produced by stimulation of the ventricular muscle in animals including the human records which have been mentioned are much notched in the initial phases These much notched curves though they do occur in records of spontaneous premature beats (Fig 17 c) are more rare than the curves with smoother upward and downward movements It may be that these premature beats with numerous notches during QRS represent beats which originate in the wall of the ventricle while the smoother curves may originate in the Purkinje system

Ventricular premature beats are frequently found to be initiated during the U wave of the preceding beat This is especially likely to be the case when there is frequent coupling It has been demonstrated that during this part of the heart cycle there is a supernormal phase of irritability and this supernormal phase has been thought to explain the tendency for ventricular premature beats to occur at this time This appears particularly to be the case when there are frequent premature beats

In precordial leads ventricular premature beats show their characteristic wide QRS group and usually show large excursions One might expect that the direction of the initial deflection of QRS in the different precordial leads would be affected by the ventricle which originated the contraction in much the same way as is found with bundle branch block Such correlations have not been investigated and it is possible that this may afford a method of localizing the origin of ventricular premature beats

Nodal premature beats A premature beat may arise in the auriculoventricular node or in the auriculoventricular bundle above its branching This gives rise to a supraventricular complex of aberrant form The P wave may be abnormal in such cases or may be normal depending upon whether the A V nodal impulse or the impulse from the sinus node initiates the contraction The impulse from the A V node will ordinarily travel back

ward into the auricles in about the same time as is consumed by the normal forward conduction so that the auricles will be stimulated by the retrograde conduction of the impulse provided that

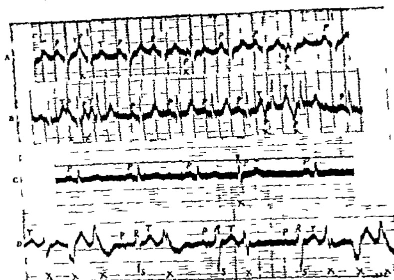


Fig 49 A Auricular premature beats from three different foci Note the different forms of the different premature P waves

B Two pairs of auricular premature beats each pair from a different focus Note that each P wave of a pair is like the other of this pair

C A premature beat arising in the A V node The P wave occurs before the proper rhythmic interval after the preceding P wave and falls between S and T of the premature beat It has a different form from the other P waves This record is by Lead 1 and in Leads 2 and 3 the P wave in this position was inverted

D Premature ventricular beats arising in several foci in succession The first three complexes have the features typical of beats starting in the ventricles and are each different in form The fourth beat is the normal for this patient It follows a P wave with a slightly prolonged A V conduction time The fifth beat is again of ventricular origin and from a still different focus The sixth beat is again supraventricular and the seventh is still a different ventricular *ectopic* beat The last three beats are of ventricular origin but repeat the form of some of the previous beats

their normal contraction has not yet started Auricular contractions due to retrograde conduction of the impulse from the A V node usually give rise to inverted P waves in Leads 2 and 3 sometimes also in Lead 1 or P_1 may have a very small amplitude as in Figure 49 C A V nodal premature beats which produce an auricular contraction by retrograde conduction are associated with an incomplete compensatory pause because the sinus

rhythm has been interrupted (see the last premature beat of Fig 46 c)

More frequently, as in Figure 50 the retrograde impulse

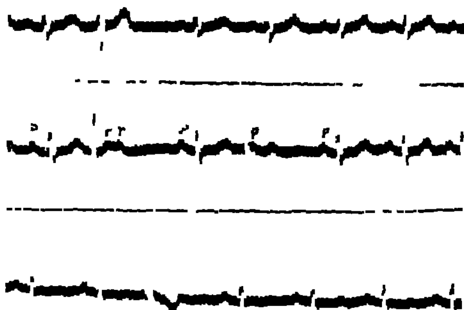


Fig 50 Auriculoventricular nodal premature beats One appears both in Lead 1 and in Lead 3 and in each the P wave is obscured by the QRS group and there is a complete compensatory pause In Lead 2 there are two premature beats and the varying degree of their prematurity causes them to appear in a different relation to the preceding T With each beat the P following the premature QRS has the normal form indicating its origin from the sinus node and the compensatory pause is complete

finds the auricles contracting in response to the sinus rhythm and so is ineffective This is recognized by the fact that the P wave occurs at the proper point in the sinus rhythm and has the normal form for the lead in question There is as in Figure 50 and as shown in Figure 46 c a complete compensatory pause with such premature beats because the sinus rhythm has not been interrupted

Irrespective of whether the retrograde impulse causes a premature auricular contraction or whether the auricles contract in response to the sinus impulse the P waves may precede the QRS

group may coincide with it or may follow it depending upon the relation of the onset of the contraction of the auricles and the ventricles When a premature beat with an aberrant supraventricular complex starts during or shortly after a normal P wave we may assume that the P R interval does not measure conduction time but only precedence of the auricular contraction Neither does the R P interval measure conduction time when a normal P wave follows the beginning of QRS but only the precedence of the ventricular contraction With inverted P waves however the R P interval indicates the time difference between the arrival of the nodal stimulus in the ventricles and auricles and is usually found to be in the neighborhood of 0 10 to 0 14 second When no P wave is found this wave has been buried in the premature QRS group and in such records a complete compensatory pause will indicate that the sinus rhythm was not interrupted by the nodal premature beat

The ventricular complex has the supraventricular form characteristic of the usual beats of the particular record although it usually shows aberration of the waves as with auricular premature beats The aberration may be quite marked in some records and is probably due to the stimulus passing into one of the bundle branches earlier than into the other

Multiple premature beats A record showing premature beats will usually be found to show but one variety These will usually have the same form throughout the record thus indicating that they arise from the same ectopic focus Interesting modifications of ventricular premature beats may occur when they come at such a time that the normal impulse and the ectopic impulse both enter different parts of the ventricles so nearly at the same time that the contraction begins at two places at once This will be recognized by a somewhat shortened P R interval being followed by a ventricular complex which seems to be a composite of that of the usual premature beat and the complex of the normal beat This condition must be distinguished from the appearance of ventricular premature beats arising from different foci In this case there will not be a short P R interval as above described

Sometimes premature beats from several different foci will be

found in one record. Figure 45 and Figure 49 A and B all show premature ectopic P waves of more than one form and Figure 49 D shows several forms of premature ventricular complexes. This indicates two or more foci of increased irritability in the auricles or the ventricles, as the case may be, and points to a more extensive and therefore more severe myocardial affection.

Premature beats from any focus may occur rarely or, as in some of the records of these figures, quite frequently. The more frequently they occur the greater the irritability at the focus. Sometimes two or three ectopic beats from the same focus may occur successively, as in Figure 49 A and B or Figure 47 B. This denotes a great local irritability and is closely allied to paroxysmal tachycardia. If different foci originate sequential ectopic beats, as in Figure 49 A, B, and D, the conditions are ripe for fibrillation of the auricles or the ventricles, as the case may be.

Sometimes, as in Figure 49 D, there will be a condition of over-irritability of several foci which may make the ventricles beat as rapidly and irregularly as they do when auricular fibrillation is present. This type of activity is in fact common in patients who show a tendency to attacks of ventricular fibrillation (page 230).

Heart Block

Heart block is an abnormality of the heart beat due to depression of the function of auriculoventricular conduction. Various sorts of arrhythmia may result (Fig. 51). If the depression of conductivity is slight it will only cause a lengthening of the time that elapses between the auricular and ventricular contractions and cannot be recognized without instrumental examination. If more marked, it leads to irregular heart action with dropped beats which may be observed by listening over the heart and by feeling the pulse. If the block is complete the heart will be regular with a slow rate, usually less than 40 per minute. Exceptionally the rate of the ventricular contractions may be much slower than this, perhaps 20 per minute for example, while in other exceptional cases it may be much faster, perhaps 70 or more per minute. A slow heart rate alone does not justify a diagnosis of heart block, nor does a rapid rate prove that it is not present.

Permanent complete heart block is due to some form of disease of the junctional tissues as are in the majority of cases the lesser grades of heart block also[1] The disease may affect either the

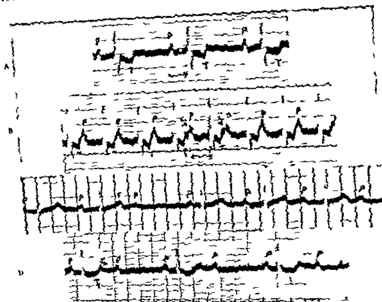


Fig 1 Auriculoventricular heart block

A Record by Le d to illustrate a prolongation of the A V *conduction time* (P R = 0 3 secs)

B A marked prolongation of the A V conduction time so that the P wave falls on the preceding T wave (P R = 0.37 secs) As the rhythm varies slightly due to sinus arrhythmia the degree of overlapping of P and T is seen to vary

C Showing the dropped beat phenomenon with increasing conduction time before the conduction fails

D The dropped beat occurs regularly after every other auricular beat so that the ventricles beat at *one* half the auricular rate

auriculoventricular node or the bundle before its branching Some authors have attempted to establish overactivity of the vagus as a cause of heart block but it is extremely doubtful if it ever produces more than a transient accentuation of a condition fundamentally due to disease A clear instance of permanent vagal complete heart block has not yet been reported although rarely the act of swallowing has been observed to cause transient

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found in one record. Figure 45 and Figure 49 A and B all show premature ectopic P waves of more than one form and Figure 49 D shows several forms of premature ventricular complexes. This indicates two or more foci of increased irritability in the auricles or the ventricles as the case may be, and points to a more extensive and therefore more severe myocardial affection.

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Fig 51 Auriculoventricular heart block

A Record by Lead 2 to illustrate a prolongation of the A V conduction time (P R = 0.28 sec)

B A marked prolongation of the A V
with the preceding T wave (P R = 0 39 s
a slight sinus arrhythmia a the degree of

C Shown the dropped beat phenomenon with increasing conduction time before the conduction fails

D The dropped beat occurs regularly after every other auricular beat so that the ventricles beat at one half the auricular rate

auriculoventricular node or the bundle before its branching Some authors have attempted to establish overactivity of the vagus as a cause of heart block but it is extremely doubtful if it ever produces more than a transient accentuation of a condition fundamentally due to disease A clear instance of permanent vagal complete heart block has not yet been reported although rarely the act of swallowing has been observed to cause transient

[1] Intraventricular block is a term applied to the results of disease affecting the branches of the bundle i.e. bundle branch block and bears no relation to the condition commonly called heart block which is auriculoventricular block

complete block with fainting dizziness or even the Adams Stokes syndrome

In Figure 51 A the P R interval was longest in Lead 2 where *it occupied 0 28 second Figure 51 B is an example of the effect* of digitalis This patient s P R interval had been 0 20 second and was increased to 0 32 second after digitalis had been given This degree of prolonged conduction would strongly suggest disease of the bundle even considering the fact that digitalis was contributing to the result

With the next grade of heart block associated with dropped beats there is occasional failure of the auricular stimulus to ar*rive in the ventricles so that a ventricular beat is lacking This* is seen in Figure 51 C where the P R interval progressively lengthens in Lead 1 from 0 22 to 0 32 or 0 10 second and the next P wave fails to be followed by a ventricular complex The gradual lengthening of the conduction time is a result of increasing fatigue of the bundle so that eventually it fails to function The next succeeding P R interval after this is again *0 22 second* This sequence of lengthening conduction time with eventual blocking of the stimulus was first described by Wenckebach and is commonly called the Wenckebach phenomenon Such dropped ventricular beats usually occur irregularly as in this record They may be very infrequent or may occur after every second or third auricular beat quite regularly for a time It is typical of this *degree of heart block for the frequency of the dropped beats to* vary from time to time so that both the rate and rhythm of the ventricles are variable

It is interesting to note the differences in the behavior of the A V conduction system in different hearts that are diseased and in the same heart at different times Sometimes there will constantly be a long conduction time as in Figure 51 B without the occurrence of dropped beats again a dropped beat will occur in the cycle *following one with a comparatively short (0 28 second)* conduction interval In one heart long records were obtained with a conduction interval of 0 60 second without a dropped beat and then suddenly the Wenckebach phenomenon appeared as in Figure 51 C with a conduction time varying from 0 28 to

0 40 second Shortly after this the heart again became regular but records were not taken

When dropped beats occur after every other auricular systole (2 to 1 block) the ventricular rate will be regular and will be just half the auricular rate so that it will usually be between 30 and 60 depending upon the rate of the auricles Record D of Figure 51 shows this condition the ventricular rate being 40 per minute and the auricular 80 with a conduction time of 0 20 second This rhythm will sometimes persist unchanged for many hours

An interesting feature of the electrical curves of heart block is that the form of the waves from beat to beat is not changed by the abnormal cardiac mechanism The P wave is usually of normal form or if abnormal it retains the same abnormality throughout The ventricular waves are often normal They may show right or left axis deviation or some other abnormality in form but with a rare exception to be detailed later they do not vary from beat to beat Note especially that even when the conduction time is varying the ventricular waves do not show *aberration* such as we find after premature auricular beats

When the P wave coincides with the larger ventricular waves it becomes submerged and may be difficult to make out Usually a comparison with other ventricular groups will enable us to note a deformity which can be identified as due to the P wave because it occurs at the proper rhythmic interval The P wave is especially difficult to make out when it falls with the T wave of the beat preceding as in Figure 51 B This superposition is rarely found in all three leads of a record so that a comparison of the time of onset of the P waves in the different leads measuring backward from the beginning of the QRS group will usually serve to decide which movement of the line of the record is P Lead I of Figure 51 B served to analyze this record by the plain separation of P and T found in this lead though the notching at the point indicated by the arrow in the lead of the figure had suggested a separation between T and the following P wave This notching occurs with variable definition because of a slight sinus arrhythmia which makes the interval between beats longer at some

complete block with fainting, dizziness, or even the Adams-Stokes syndrome.

In Figure 51 A the P-R interval was longest in Lead 2 where it occupied 0.28 second. Figure 51 B is an example of the effect of digitalis. This patient's P-R interval had been 0.20 second and was increased to 0.32 second after digitalis had been given. This degree of prolonged conduction would strongly suggest disease of the bundle, even considering the fact that digitalis was contributing to the result.

With the next grade of heart block associated with dropped beats there is occasional failure of the auricular stimulus to arrive in the ventricles so that a ventricular beat is lacking. This is seen in Figure 51 C where the P-R interval progressively lengthens in Lead 1 from 0.22 to 0.32 or 0.40 second and the next P wave fails to be followed by a ventricular complex. The gradual lengthening of the conduction time is a result of increasing fatigue of the bundle so that eventually it fails to function. The next succeeding P-R interval after this is again 0.22 second. This sequence of lengthening conduction time with eventual blocking of the stimulus was first described by Wenckebach and is commonly called the Wenckebach phenomenon. Such dropped ventricular beats usually occur irregularly as in this record. They may be very infrequent or may occur after every second or third auricular beat quite regularly for a time. It is typical of this degree of heart block for the frequency of the dropped beats to vary from time to time so that both the rate and rhythm of the ventricles are variable.

It is interesting to note the differences in the behavior of the A-V conduction system in different hearts that are diseased and in the same heart at different times. Sometimes there will constantly be a long conduction time as in Figure 51 B without the occurrence of dropped beats; again a dropped beat will occur in the cycle following one with a comparatively short (0.28 second) conduction interval. In one heart long records were obtained with a conduction interval of 0.60 second without a dropped beat and then suddenly the Wenckebach phenomenon appeared as in Figure 51 C with a conduction time varying from 0.28 to

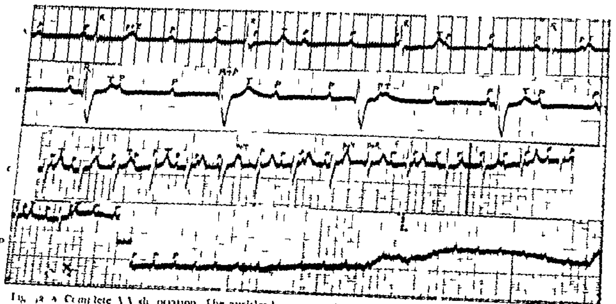


Fig. 18. A. Complete A-V dissociation. The auricles beat regularly 80 times a minute and the ventricles regularly at times a minute. Note the auricular P wave.

B. Complete A-V dissociation, the ventricular complexes showing an abnormal form. Note the auricular P wave.

C and D. A continuous record of a patient who was having Adams-Stokes syndrome with convulsive attacks. The ventricular beats stopped at X, the auricles continuing to beat to the end of the strip. The movement of the record at Y was due to the onset of a convulsion fourteen seconds after the last ventricular systole.

times than at others and in the longer pauses separates P from the preceding T wave

The next increase in the severity of heart block leads to a rather *uncommon condition There is an incomplete dissociation between the auricles and the ventricles* so that the stimulus comes through to the ventricles only occasionally For the other beats *the ventricles contract with a slow regular rhythm which arises* in a portion of the auriculoventricular system below the lesion that interferes with the function of the bundle If this is above the division of the bundle into right and left branches *this fact* will be recognized by the ventricular complex having the supraventricular form though such complexes may be aberrant due to a diminished facility of conduction into one or the other branch of the bundle In other cases the complexes have the appearance of ventricular ectopic beats which is usually due to the rhythm originating in some portion of the right or left bundle branch A similar electrocardiogram will be obtained if the impulse arises in the A V bundle above the branching and is blocked in one branch This mechanism is indistinguishable from the last one mentioned

A much more common manifestation of block than that last mentioned is a complete dissociation between auricles and ventricles as shown in Figure 52 A The P waves are seen to be regular at a rate of 89 per minute the ventricular waves being 26 per minute and also regular The ventricular waves in this figure have the characteristic supraventricular form Figure 52 B shows complete heart block with the ventricular complexes of abnormal form and therefore probably not of a supraventricular origin The pathological changes causing complete *heart block often* extend into one bundle branch and in such cases the ventricular rhythm may be originated in this branch at a point beyond the lesion giving rise to a record like that of the figure A similar record might result if a patient with bundle branch block developed complete A V block also The supraventricular impulse would then produce a bundle branch block complex instead of a normal one

Occasionally with complete heart block there will be ventricular complexes of the bundle branch block type which will vary

Patients with complete heart block may also suffer from Adams Stokes attacks because of the sudden onset of ventricular fibrillation. This as has been shown by Schwartz and Jezer may last from a second or so to as long as five minutes. Shorter attacks are not usually associated with unconsciousness but during the longer ones the patient becomes unconscious and shows typical convulsive seizures. A continuous record of a part of a short attack lasting 24 seconds is shown in Figure 62 which was kindly furnished by Dr S. Schwartz. These authors have pointed out that patients subject to syncopal attacks due to this mechanism are liable to show certain premonitory alterations in the cardiac mechanism. First there is an increase in rate of the basic idioventricular rhythm. Then ectopic premature ventricular beats appear and may come singly at first and later in groups. This latter mechanism gives way to short or long runs of the recurrent oscillations which are due to ventricular fibrillation. If these runs last for as much as 30 to 40 seconds typical convulsive Adams Stokes attacks supervene. Such phenomena do not occur when Adams Stokes attacks are due to ventricular asystole as in Figure 52 so it follows that if premature beats are found to occur frequently in patients with complete heart block and Adams Stokes attacks it may be presumed that the attacks are due to ventricular fibrillation rather than to asystole.

When incomplete heart block is present Adams Stokes attacks may occur by a quite different mechanism. A partial block suddenly becomes complete and a period of from 5 to 8 seconds may elapse before the auricular stimulus again passes to the ventricles or the ventricles take on the function of originating a rhythm [illegible]
co [illegible]
lo[illegible] [illegible]ousness

Certain records with complete heart block appear to show the occasional conduction of an impulse in a retrograde direction even though forward conduction from auricles to ventricles does not take place. At times there is apparently a state of unusual facility of conduction set up by the recent occurrence of the penetration of a ventricular impulse backward into the region of depressed conduction or by the successful passage of an auricular

in form from beat to beat There is usually also a slight coincident irregularity in the ventricular rhythm These variations in form and in rhythm are due to a varying site of the impulse formation within the A V bundle and its branches Premature beats also occasionally arise in the ventricles when complete heart block is present interrupting the slow regular rhythm

Auricular fibrillation may be present coincident with complete heart block as in Figure 60 B This will make no difference to the ventricles which are removed from auricular influence by the block and which will continue to beat slowly and regularly unless there should be premature beats or a varying site of the origin of the stimulus The effect of partial heart block upon the heart with auricular fibrillation will be discussed under the latter heading It causes a slow but irregular ventricular action

The electrocardiographic records are able to cast a very interesting light upon the mechanism which precedes and accompanies attacks of Adams Stokes syndrome C and D of Figure 52 are parts of a continuous record by Lead I showing the heart action in one of these cases The condition at the beginning of record C was complete heart block with an auricular rate of 96 and a ventricular rate of 54 per minute The ventricular complexes first were of the left ventricular type probably due to modification of the supraventricular impulse by right bundle branch block Later the impulse to the right ventricle was less delayed and the complexes resembled the normal ones of this case There were frequent variations from beat to beat however Suddenly at X the ventricles ceased to beat—their automatic center failed to function—the auricles meanwhile continuing to beat as before After 15 seconds had elapsed the patient had a general tonic convulsion during which the ventricles again started to beat as at the beginning of record C and the cycle was repeated This patient had convulsion after convulsion about six times an hour The attacks were always preceded by cessation of the heart sounds and the ventricular beats always resumed It is probable that the irritability of their automatic center was enhanced by the asphyxia to a point at which it again gave rise to contractions

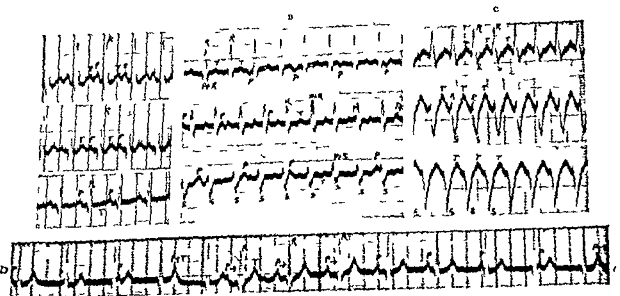


Fig. 3. Varieties of tachycardia.

A. Sinus tachycardia. Rate 1[illegible]0 per minute.

B. Tachycardia of [illegible] origin (A-V node) combined with complete A-V dissociation. Auricular rate 103 per minute; ventricular rate 180 per minute.

C. Tachycardia of ventricular origin (left ventricle). The rate is 2[illegible] per minute. [illegible] cannot be discovered.

D. Tachycardia originating in the A-V node, but having a rate only slightly faster than the auricle. Auricular rate 8[illegible] per minute, ventricular rate [illegible] per minute. For three heart cycles in the center of the record the auricular beat is followed by the ventricular in the normal manner, proving that the A-V conduction function is not disturbed. The first ventricular beat [illegible] at a shorter interval and probably originates from the A-V node, as do the later ones. Note the aberrant ventricular complexes of the nodal rhythm. They have no S wave and [illegible] not so high.

impulse forward through it This may give rise to a single functioning of the auriculoventricular tissue or to periods of variable duration when each auricular impulse is conducted This latter phenomenon has been ascribed to a supernormal recovery phase being set up in the diseased tissue of the auriculoventricular bundle in the sense that this term was used by Adrian and Lucas that is that there is a short period during the recovery from previous activity during which period the tissue becomes hypersensitive to new stimuli

TACHYCARDIA

Sinus tachycardia A tachycardia which might be called physiological in that it is a simple acceleration of a normally contracting heart is recognized from the electrocardiogram by the P wave and the ventricular complex having the normal form for the patient in question or having only such slight changes as may be the result of the rapid rate These changes are a short duration of P R of QRS and of S T and a slight reduction in the size of the waves A lower limit for the rate of simple tachycardia has been arbitrarily designated as 100 per minute by the Criteria Committee of the New York Heart Association Figure 53 A is a record of such a tachycardia the rate of auricles and ventricles being 150 per minute

The rate of these physiological tachycardias increases gradually at their beginning and declines gradually when the rate slows In this they are distinctly contrasted to what might be called the pathological tachycardias which are paroxysmal in type starting and stopping abruptly the rate remaining practically constant during their course The rate of a physiological tachycardia is seldom found to be above 150 per minute while the paroxysmal tachycardias usually though not always have much higher rates Any rate over 150 per minute should be regarded as probably due to paroxysmal tachycardia unless a record has proved it to be physiological or unless exophthalmic goiter is present The latter may cause very rapid physiological heart rates as well as abnormal rhythms

Paroxysmal tachycardias due to the regular activity of ectopic foci may have a rate as slow as 100 per minute or less or as fast
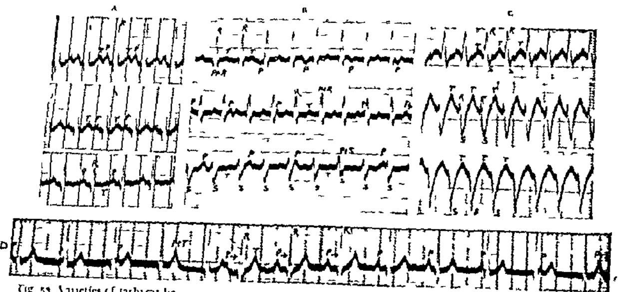


Fig. 55. Varieties of tachycardia.

A. *Sinus tachycardia.* Rate [illegible] per minute.

B. Tachycardia of nodal origin (A-V node) combined with complete A-V dissociation. Auricular rate 108 per minute; ventricular rate 180 per minute.

C. Tachycardia of ventricular origin (left ventricle). The rate is [illegible] per minute. P waves cannot be discovered.

D. Tachycardia originating in the A-V node but having a rate only slightly faster than the auricles. Auricular rate 81 per minute. Ventricular rate [illegible] per minute. For three heart cycles in the center of the record the auricular beat is followed by the ventricular in the normal manner, proving that the A-V conduction function is not disturbed. The next ventricular beat follows at a shorter interval and probably originates from the A-V node, as do the later ones. Note the aberrant ventricular complexes of the nodal rhythm. They have no S wave and T is not so high.

as 210 per minute or more in different cases They are the result of the activity of an ectopic area of increased irritability just as are premature beats and are, in effect, a rapidly recurring series of premature beats which takes control of the heart rhythm Paroxysmal attacks of rapid heart action which are due to auricular flutter or auricular fibrillation although actually associated with tachycardia are *not to be grouped with the attacks here designated*

The point of origin as with premature beats, may lie in any part of the auricles or ventricles or in the A V junctional tissue The attacks may last for a period of only a few heart cycles or for several minutes or hours depending upon how long the irritability of the focus is so increased that it continues to originate beats Figure 49 B shows two successive premature auricular beats *which from the similarity of their P waves can be considered to* come from the same focus Figure 47 B shows two successive premature ventricular beats from the same focus From a physiological point of view two successive premature beats constitute a short attack of paroxysmal tachycardia but attacks to be clinically recognizable must last for several seconds at least

At the end of a paroxysm of tachycardia the ectopic focus suddenly ceases to produce stimuli and there is a pause which is *usually longer than the interval between beats of the normal* sinus rhythm—sometimes as long as several of these intervals The sinus node may then begin to function and P waves make their appearance which are followed by ventricular complexes At times the auricular contractions do not appear for a few seconds and one or more ventricular beats of the nodal type may appear irregularly the mechanism being in effect ventricular escape Sooner or later however the normal rhythm is resumed

Auricular tachycardias nearly always show a P wave of an obviously abnormal form and always one of an abnormal form for the individual in question The focus which originates the impulse *is ectopic so that the auricles contract in an abnormal* manner and produce a P wave which is abnormal for this person In Figure 54 A the auricular wave appears as a notch upon the descent of the T wave because of the rapid heart rate of 195 per minute Thus P wave does not appear very abnormal on first sight

but if contrasted with that of Figure 54 B which was obtained after the paroxysm had subsided it will be seen to be different Notice also that the ventricular waves of Figure 54 A are dis

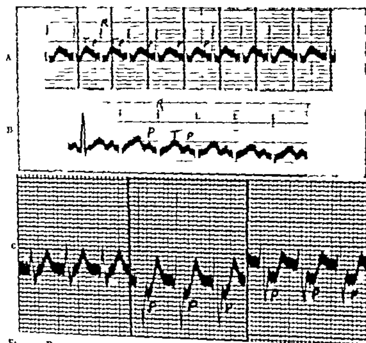


Fig 54 Paroxysmal tachycardia
Paroxysmal auricular tachycardia with auricular rate 19 per minute
B Later record of the same patient by the same lead The rate is now 14 per minute a simple tachycardia Note the different form of the T wave in these two records

Paroxysmal tachycardia originating in the A V node Rate 160 per minute The P wave can be seen as an inverted notch at the beginning of the S T segment in Leads and 3 It follows the beginning of QRS by 0 10 second

tinctly aberrant as compared with those of the normal rhythm in Figure 54 B lacking the tiny S wave and showing a different form of the S-T segment and of T During paroxysms of auricular tachycardia the ventricular waves are usually found aberrant just as they are after isolated auricular premature beats

Figure 53 B is a record of a *nodal tachycardia* the impulse originating in the A V node In this record there is also a complete dissociation between the auricles and the ventricles the former beating regularly 108 times per minute and the latter 180 times

as 210 per minute or more in different cases They are the result of the activity of an ectopic area of increased irritability, just as are premature beats, and are in effect a rapidly recurring series of premature beats which takes control of the heart rhythm Paroxysmal attacks of rapid heart action which are due to auricular flutter or auricular fibrillation although actually associated with tachycardia are not to be grouped with the attacks here designated

The point of origin as with premature beats may lie in any part of the auricles or ventricles or in the A V junctional tissue The attacks may last for a period of only a few heart cycles or for several minutes or hours depending upon how long the irritability of the focus is so increased that it continues to originate beats Figure 19 B shows two successive premature auricular beats which from the similarity of their P waves can be considered to come from the same focus Figure 17 B shows two successive premature ventricular beats from the same focus From a physiological point of view two successive premature beats constitute a short attack of paroxysmal tachycardia but attacks to be clinically recognizable must last for several seconds at least

At the end of a paroxysm of tachycardia the ectopic focus suddenly ceases to produce stimuli and there is a pause which is usually longer than the interval between beats of the normal sinus rhythm—sometimes as long as several of these intervals The sinus node may then begin to function and P waves make their appearance which are followed by ventricular complexes At times the auricular contractions do not appear for a few seconds and one or more ventricular beats of the nodal type may appear irregularly the mechanism being in effect ventricular escape Sooner or later however the normal rhythm is resumed

Auricular tachycardias nearly always show a P wave of an obviously abnormal form and always one of an abnormal form for the individual in question The focus which originates the impulse is ectopic so that the auricles contract in an abnormal manner and produce a P wave which is abnormal for this person In Figure 54 A the auricular wave appears as a notch upon the descent of the T wave because of the rapid heart rate of 195 per minute This P wave does not appear very abnormal on first sight

minute as in Figure 53 D The condition is then properly described as a *nodal rhythm* not as a nodal tachycardia There is also the rather rare but characteristic slow nodal rhythm previously described with inverted P waves plainly seen after the QRS group The Q P interval in such records may be as much as 0 18 second

Figure 53 C shows a tachycardia which arises in the left ventricle a ventricular tachycardia The P waves are entirely lost in the large excursions of the ventricular waves which have the typical wide notched QRS and the large T wave directed opposite to the chief deflection of the QRS group indicating an origin in the ventricular tissues or the left branch of the A V bundle They resemble the complexes of the premature ventricular beats of Figure 48 C which arise in the left ventricle except that they have a larger R_1 and smaller S_1 They more closely resemble the complexes of Bayley s type 4 right bundle branch block seen in Figure 96 A In other records of ventricular tachycardia as in Figure 6 the P waves may be discerned occurring in a slower independent rhythm as they do in the record of nodal tachycardia in Figure 53 B Ventricular tachycardias are much less frequent than those of auricular or nodal origin Paroxysms also are [illegible]

[illegible]

to o [illegible] are either in the bundle branch tissue or in the muscle of the ventricle

Parasystole An example of independent parasystolic rhythms may be seen in Figure 53 D where the A V node and the sinus node each have an independent rhythm competing for the control of the heart beat (see also Fig 43 E) Here the auricles occasionally succeed in producing a ventricular beat which is premature in respect to the A V nodal rhythm governing the heart rate This happens whenever the auricular impulse does not find the ventricles refractory In this case the auricles are protected from stimuli from the A V node by a blocking of retrograde impulses while there is no forward block to prevent the auricular impulse from being effective The term *interference dissociation* has also been applied to this phenomenon

Sometimes a nodal tachycardia may have a rate not much faster than the auricles and whenever the impulse from the auricles *finds the ventricles in the diastolic resting state* it forces a contraction which slightly disturbs the regularity of the ectopic rhythm This condition has been erroneously termed *ventricular escape* and is illustrated in Figure 53 D where the auricles are 84 and the ventricles 90 per minute The ventricular complexes have a typical supraventricular form showing that the impulse arises from the A V junctional tissues The P waves are seen to bear no constant relation to the ventricular waves Heart block *is not present in this record for at the points indicated by the* arrows the auricular impulse has caused a ventricular contraction after a normal conduction time All other impulses from the *auricles found the ventricles contracting and therefore refractory* to stimulation Note that the ventricular complexes of the nodal rhythm are aberrant as compared with those which are due to the normal impulse They lack an S wave and the T is not so high

The ventricular complexes of Figure 53 B are also aberrant as compared with those which were found between the attacks the latter showing less left axis deviation a briefer QRS group and an inverted T_3 We recognize this rhythm as being *nodal* by the fact that the ventricular complexes have a typically supraventricular form with a duration of the QRS group within normal limits *The P waves of this record are abnormal in having* an inversion of P_1 This would indicate that the auricular rhythm was ectopic arising from an abnormal auricular focus

In other records of nodal tachycardia as Figure 54 C there is an inverted P wave *following the beginning of QRS* by an interval of from 0 10 to 0 14 second In such cases the impulse originating in the node has passed into the ventricles and backward into the auricles at the same time as has been described when *discussing nodal premature beats In other records* no P wave can be made out and in such records it may have coincided with QRS

In certain other cases *the rate is not excessive and the rhythm* is controlled by the A V node The rate may be less than 100 per

flutter may be transient or may be permanent lasting sometimes for many months or even years Records are seen in Figure 55 A B and C The typical feature is the continual up and down wavy movement of the base line at a rate of about 300 per minute each peak being about 1/5 second from the next This wavy line is due to the auricular activity and has the ventricular waves superimposed upon it It is usually best seen in Leads 2 and 3 In Lead 1 the waves due to the auricles are often very small and difficult to make out as in Figure 55 A and C It is often difficult to say which is the zero level of these records Most records of auricular flutter show this practically continuous movement of the base line but with slower rates i e below 255 it seems as if the zero level were midway between the peak and the trough of these waves the deflection being first sharply downward and then rising rather sharply above zero to return to it again before the next wave starts This can be observed in Figure 55 C and less plainly in Figure 55 A Records which have caught the *onset* or cessation of auricular flutter bear out this statement for the zero level of the record during normal rhythm lies midway between the peak and trough of the wavy movement due to the flutter Different patients have waves of different form resulting from auricular flutter probably depending upon the special path of the contraction in the auricular muscle Some records show the flutter waves *more* plainly in Lead 1 and less plainly in Lead 2 It is rare for records to show the flutter waves poorly in Lead 3 In certain records as in Figure 55 D the form of the flutter waves is more rounded and resembles somewhat the usual P wave except that its duration is much greater

The auricles appear to have little or no diastolic period one electrical wave continuing on into the next It must be that some part of the potential of these waves is produced during the diastole of an appreciable part of the auricular wall for the polygraph shows a succession of pressure waves due to successive auricular co[illegible] the polygraph waves is d[illegible] must mean diastole o[illegible] major part of the muscle of the auricle The cause of the electrical deflection during this diastole must involve the activity of only a part of the auricular muscle

The rate of the auricular waves varies in different records from

Occasionally premature beats have been found to occur in the course of a dominant rhythm these beats occurring with a definite rhythm of their own This has been considered to in dicate that they arise from a separate individual center Such a paarrhythmia can only be recognized by careful study of a long strip of the record because the parasystolic rhythm usually is interrupted by beats of the normal rhythm as in Figure 46 H The recognition of this condition is further complicated because certain stimuli of the pararrhythmia fail to produce ventricular beats because of finding the ventricles refractory contracting in response to the dominant rhythm It is necessary to suppose that the secondary center is protected against excitation by the normal beats through a mechanism which has been called protective block Otherwise the pararrhythmia would be interrupted

In most such cases the secondary rhythm has a rate slower than the sinus rhythm which is controlling the heart beat and usually the focus is within the ventricles so that the effect is that of fre quent ventricular premature beats Similar auricular foci have been observed This theory has been particularly elaborated by Rothberger and his associates

Other records have shown the ectopic rhythm occurring at a rate faster than the sinus rate When this condition is present there is also the constant activity of a secondary pacemaker com peting with the sinus rhythm for control of the heart beat Cer tain records have been thought to indicate the action of more than one of these secondary pacemakers With rhythms faster than the sinus rhythm one must assume a blocking of certain of the stimuli from the secondary center so that only occasionally does the ectopic ventricular stimulus break through this block and produce a ventricular beat The pararrhythmic center also must be protected against excitation by the normal beat through the previously mentioned mechanism of protective block In spite of the elaborate theoretical basis which is necessitated for the assumption of pararrhythmia there are numerous records available which seem to indicate that such a mechanism can exist

Auricular Flutter

Paroxysmal attacks of rapid heart action may occur by a still different mechanism called auricular flutter Attacks of auricular

as low as 178 to as high as 350 per minute In Figure 55 it is 288 per minute for record A and 291 for B and C The ventricles respond to every second auricular impulse in records A and B and irregularly to the second or third impulse in record C

Figure 55 D shows the result of vagus stimulation upon the A V conduction in the patient who gave record B Pressure upon the carotid sinus in the neck on either right or left side *rarely fails to slow the ventricular rate* when flutter is present though the effect usually is only brief Figure 55 D was obtained immediately after 55 B was recorded but during pressure on the left carotid sinus The effect of vagus stimulation upon the F waves is very slight and transient consisting of a very slight change in the rate This change usually lasts only a second or two but is definitely measurable in this record and in all the records so far published in medical literature showing the effect of vagus stimulation in the presence of flutter It can be seen in Figure 55 D It is a variable effect sometimes slowing and sometimes quickening the rate by a few beats per minute Lewis has noted a similar variable effect upon the auricular rate of flutter when the patient changes from the standing to the lying position

A 2 1 relation of auricular and ventricular waves is the usual thing with auricular flutter but this depends upon the functional condition of the A V bundle and 3 1 or 4 1 or irregular ventricular responses are commonly found Cases showing 1 1 response have been reported and the records published The rate in these records has usually been slower than 250 per minute

The 2 1 relation of auricular rate to ventricular is not evidence of subnormal function of the A V bundle with these rapid rates for the impulse from the second auricular beat arrives while the ventricles are still contracting in response to the impulse from the first auricular beat so that it finds the ventricles refractory to stimulation If two or more auricular beats fail however one of the impulses must have been blocked in the A V system The blocking shown in Figure 55 C was due to digitalis

The *physiology* of auricular flutter is fundamentally quite different from that of auricular tachycardia flutter being a more marked disturbance of auricular function Tachycardia tends to

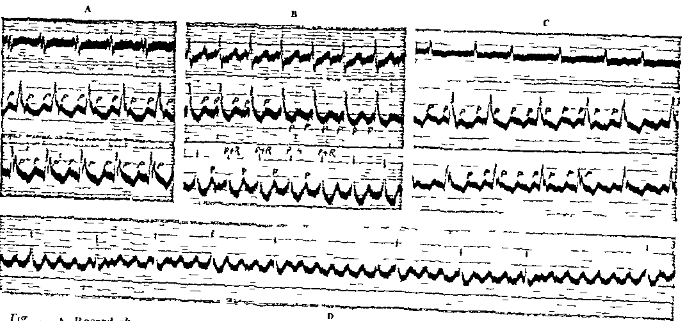


Fig [illegible]. A Record showing auricular flutter with 2:1 relation of auricular and ventricular waves. Auricular rate 288 per minute. The QRS group is notched and slurred.

B Another record of auricular flutter showing 2:1 relation of *auricles and ventricles*. Auricular rate 294 per *minute*.

C Later record from the patient who gave record A showing a variable 2:1 or 3:1 auriculoventricular relation. Auricular rate 288 per minute. The auricular waves are smaller in this record. The patient was somewhat under the influence of digitalis.

D Record by Lead 3 from the patient who *gave record B to show* the result of pressure upon the carotid sinus. This record was taken immediately after record B.

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The *physiology* of auricular flutter is fundamentally quite different from that of auricular tachycardia flutter being a more marked disturbance of auricular function Tachycardia tends to

have lower auricular rates, rarely above 215 per minute while auricular flutter tends to higher ones usually above 240 per minute The similarity between all curves of auricular flutter as

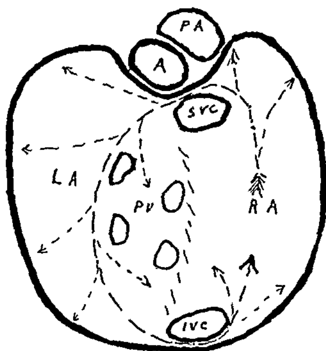


Fig 56 Diagrammatic view of the auricles from behind to indicate the usual course of the circus contraction of auricular flutter The central circus contraction *indicated by the line of dots and dashes is seen to surround the superior vena cava* (*S V C*) pulmonary veins (*P V*) and inferior vena cava (*I V C*) There is a gap of quiescent muscle between the tail and the head of the contracting portion *which may be greater or less than the gap here indicated The contraction pro*gresses in the direction indicated and spreads to the remainder of the auricular muscle as shown by the arrows radiating from the central path

contrasted with the variable appearance of the auricular waves in tachycardia is additional evidence of a physiological difference between these two mechanisms and in favor of a common mechanism for all cases of flutter

Lewis has put forward an hypothesis as to the mechanism of auricular flutter based upon *the observations of Mines and* Garrey and elaborated by experiments of his own It is in such excellent *agreement with all that had been brought forward pre*viously that it seems undoubtedly correct Flutter he believes *is brought about by a change in the physiological condition of*

the muscle that *delays the rate at which the contraction passes through it* Thus the contraction wave having swept across a part of the wall of the auricle and returned to its point of origin finds the contraction process completely passed away there so that the region is again capable of contracting This it does and the contraction spreads again along its former path and again on returning to the starting point this has ceased to be refractory is again irritable and again contracts There is thus formed a ring around which the contraction wave is coursing Behind the contracting portion of the ring is an area which is relaxed and which enters into contraction as the front of the wave proceeds (Fig 56)

This process was described as a *circulating rhythm* by Mines who said Such a wave (of contraction) runs round the ring (of auricular muscle of the ray) sufficiently slowly for the refractory phase to have passed off in each part of the ring when the wave approaches it Thus the wave circulates and may continue to do so Garrey introduced the term circus contraction which is so descriptive that it seems worth retaining

In most of the dogs which he investigated Lewis found that the path of the circus contraction was around the entrance into the auricles of the superior vena cava right pulmonary veins and inferior vena cava as shown in Figure 56 which is modified from one of his illustrations In others it surrounded only the superior cava and the right pulmonary veins and in one dog it appeared to surround the left A V orifice From the central path of the circus contraction the other parts of the auricles are involved by a radial spreading of the contraction as shown in the figure and with each circus contraction this radial spreading is repeated The radial paths like the central circus are always the same and thus the auricular waves are always the same

Lewis also observed in dogs a condition which he called impure flutter This is characterized in its lesser grades by slight irregularity in the form of occasional flutter waves As the condition becomes more marked there may be a slight variation in the form and rhythmicity of groups of flutter waves The pre

dominant flutter rhythm is not permanently disturbed however, and continues after the impure flutter ceases He suggests that *this disturbance is due to temporary variations in the paths of*

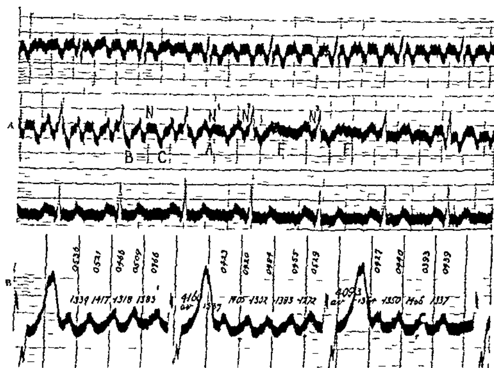


Fig 57 Impure flutter

A Three lead from the case reported by Cookson and Clark Kennedy Lead 1 shows impure flutter during the first third with a rate of 412 per minute The rest of this lead shows pure flutter at the same rate During the first part of Lead 2 alternate flutter waves show similar differences of form until the point N after which there is a somewhat irregular variation in these waves In the center of the record the waves E and F seem to mark two flutter waves of a more usual type the rate being half of the previous rate Toward the end of the record the rapid flutter waves reappear without showing the alternating peculiarity of the first part Lead 3 shows pure flutter with a rate of 206 In all three leads the ventricles are regular at a uniform rate of 103 (Cookson and Clark Kennedy *Heart* 16 10, 1932)

B Lead 2 from one of Lewis experiments on dogs showing impure flutter with distortion of the auricular complexes The figures written horizontally are measurements of the intervals separating the auricular complexes Note that the *auricular complexes are not uniform in shape the inconstancy of the notch* marked with the + sign may be noted especially (Lewis T *Heart* 8 293 1918 20)

radial spreading of the contraction or to temporary variations in the central path of the circus contraction This condition constitutes the borderline between flutter and fibrillation and is usually associated with a more rapid than usual rate of the flut

ter movement It is a rare condition in clinical records but one is seen in Figure 57 A Figure 57 B is one of Lewis experimental records to show another type of variation of the flutter wave The notch indicated by the + mark varies from cycle to cycle as does the form of the peak but the fundamental rhythm is still maintained as shown by the horizontally written measurements of the F – I interval above the waves

Auricular Fibrillation

Paroxysmal attacks of rapid ventricular rate may also be caused by the sudden inception of what is called fibrillation of the auricles But fibrillation may also be a permanent condition and in fact usually is so In either case the ventricles beat irregularly Electrocardiograms of this condition are shown in Figure 58 the typical characteristics being (1) absence of a wave of constant form which might be due to auricular contraction preceding each ventricular wave (2) in the interval between the ventricular complexes a series of wavelets marked *f f f* which vary in height and width (i e rate) Certain of these waves may closely resemble a P wave and may occur at the proper interval before some of the ventricular complexes but if the record is examined at large it will be found that at best this is only an occasional occurrence as other waves will have a slightly different form or will occur at a different interval before the ventricular complex

Figure 58 A is from the patient who had previously given records A and C of Figure 57 and it is seen that the regular waves of auricular flutter have given way to a more rapid series of waves which are not so large and which diminish from time to time until the movement of the line is very slight It will be noted that there is no significant difference in the ventricular waves In different patients the rate of the auricular waves varies from 350 to 500 per minute but the characteristic feature is that they are constantly changing form and that they tend alternately to increase and decrease in size Figure 58 B shows a record with extraordinarily plain auricular waves in Leads 1 and 3 which might suggest a diagnosis of auricular flutter These waves

appear to be regular in parts of the record but if carefully measured are found not to be so. Moreover they vary in *height* and *form* and in parts of the record can be seen to disappear almost

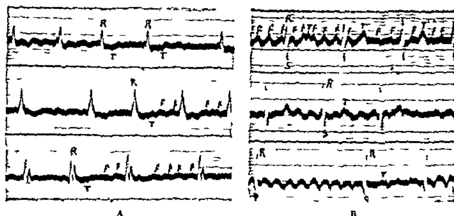


Fig 58 A Auricular fibrillation. The small wavelets *f f f* are due to the auricular activity. The ventricular waves occur irregularly and are not preceded by any wave of constant form which might be a P wave. Note the slurring or notching of the QRS group in three leads.

B Auricular fibrillation with large auricular waves. The ventricular complexes show right axis deviation of QRS.

entirely. This is in strong contrast to what is found with auricular flutter whose waves measure equal over long intervals and are of uniform height and form. A more usual record of auricular fibrillation is seen in Figure 58 A. These two records show a rate of about 100 per minute for the slowest fibrillation waves. Other records may show rates as high as 500 per minute.

The height of the waves due to auricular fibrillation seems to depend upon the same factors that influence the height of P (page 111). At least they are found large most often in records from hearts with mitral stenosis (auricular hypertrophy) and small most often in records from hearts with diffuse myocardial disease and with the myocardium in poor condition. Figure 59 B is from a case of diffuse disease and the record shows only small fibrillation waves varying in size and sometimes disappearing altogether for long stretches.

During fibrillation of the auricles there is no coordinate contraction of the muscle fibers of these chambers sufficient to cause a pressure variation within them. This fact was apparent in

polygraphic records and led to an hypothesis of auricular paralysis Individual fibers or groups of fibers continue to contract but the *shortening* of some fibers is neutralized by the synchronous

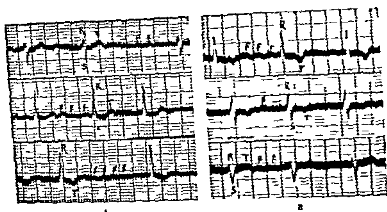


Fig 9 A Auricular fibrillation with small excursions of the auricular waves The ventricular complexes show right axis deviation of QRS
B Auricular fibrillation with very small auricular excursions The ventricular complexes show left axis deviation of QRS and a downward T wave in Leads 1 and 2

relaxation of others so that the net result is a practically constant size of the auricular wall

Lewis has pointed out that there is a relation between the auricular physiology in flutter and in fibrillation In each case there is a *circus contraction* but in fibrillation its path is not always the same There is an increase in the rate of *progress* (spreading) of the contraction in the central ring a shortening of the path of the circus contraction and a shortening of the duration of the contraction (refractory period) of the individual muscle fibers This leads to continual variations in the length and course of the central path and therefore to variations in the radial paths by which the contraction spreads from the central ring to the outlying parts of the auricular muscle (Fig 56) This theory explains why the fibrillation waves vary progressively in a given record and goes far to make clear many other clinical and experimental features of auricular fibrillation

Several authors have observed in experimental animals what they considered to be a combination of auricular flutter and

auricular fibrillation In addition to the rapid series of contraction waves passing over the auricular wall which can be plainly seen when the auricles are in flutter there were simultaneous fine fibrillary movements more especially observed along the auriculoventricular groove and in the appendices It may be that this condition is responsible for the large well defined fibrillation waves of such records as Figure 58 B and 60 A which at times strongly suggest the wavy line of flutter These records may be referred to as showing coarse fibrillation waves but it is clear that they indicate fibrillation and not flutter We must distinguish impure flutter (page 157) from this condition by the fact that the waves are quite regular and are continuous throughout not showing the phasic increase and decrease of amplitude which is found with fibrillation

The *ventricular response during auricular fibrillation* depends upon the number of effective impulses which are passed to the ventricles by the A V system The functional integrity of this system is the most important factor in determining the ventricular rate though it is not impossible that there may be other factors There may be qualitative differences in different auricular stimuli passing from the auricles to the A V node so that only certain stimuli are effective It may be that impulses come to the A V node irregularly or there may be a summation of small stimuli in the node until the level of impulse formation is reached

The usual thing when auricular fibrillation sets in is to find the ventricular rate as in Figure 60 A very close to the maximum which could be attained considering the fact that the ventricles are refractory to a second stimulus until the T wave is completed In this record the rate is 186 per minute and the irregularity very slight It would scarcely be appreciated by the finger on the pulse and can be recognized only by very careful auscultation at the apex Pressure upon the carotid sinus in the neck always will produce a brief slowing of the ventricular rate and make the irregularity more evident by blocking some of the impulses from the auricles Digitalis slows the ventricular rate by its ability to increase the activity of the vagus and disease of the node or main stem of the bundle also reduces this rate

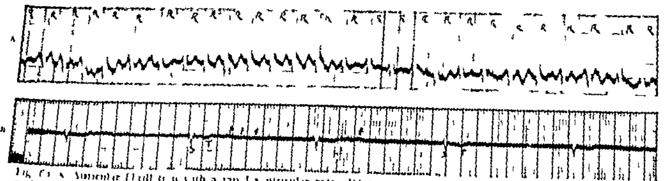


Fig. [illegible]. A. Auricular flutter with a rapid auricular rate—[illegible] per minute. B. Auricular fibrillation combined with complete heart block. The ventricles are perfectly regular at [illegible] per minute. Both the auricular and ventricular waves were as small in the other leads as in this one.

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mature ventricular beats but in sections C and D there are waves which have a duration varying from 0.24 to 0.32 second which are so unusual in their configuration that they scarcely resemble

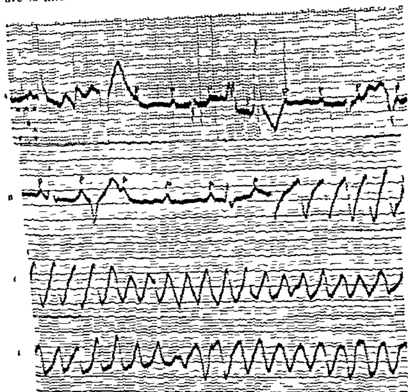


Fig. 61. Illustration of the onset of ventricular fibrillation.
A. Shows the frequent ventricular ectopic beats which occur in the prefibrillation phase.
B. The end of a long period of coupled beats and the beginning of ventricular fibrillation.
C and D are continuations of B and show the variations in the fibrillation waves. The attack terminated with recovery after three minutes. (Records B, C and D were made available through the kindness of Dr. Sidney P. Schwartz.)

the other waves. This series of waves is due to ventricular fibrillation. The variations in form and duration are quite analogous to those observed in the waves of auricular fibrillation though the waves and their duration are much larger. These changes in the form of the waves as the attack begins and progresses are usually evident in records of attacks. They have been particularly

With rates between about 120 per minute and 70 or 75 per minute the irregularity is usually more marked than when the rate is above or below these limits Some patients continually maintain a ventricular rate of 75 or under without taking any medication Such patients have either a vagotonia or an impairment of the A V system due to disease for without one or the other they would have a ventricular rate of 90 to 100 or more which is the lowest rate that a patient unaided by digitalis seems able to attain through the normal activity of the vagus

When auricular fibrillation is present and the ventricles are perfectly regular as seen in Figure 60 B we know that complete heart block is present This would be so even if the ventricular rate were faster for if the A V system is capable of functioning at all it will transmit impulses irregularly when the auricles are fibrillating

Aberrant ventricular complexes like those found with auricular premature beats are especially common with auricular fibrillation when the rate is rapid as are also premature beats of ventricular origin

Ventricular Fibrillation

Just as the auricular muscle goes into fibrillation so the ventricular muscle may take on this same incoordinate activity under certain circumstances It is probable that ventricular fibrillation occurs much more frequently than is observed for if it lasts more than a few minutes or so the patient cannot live If the ventricles do not beat coordinately they will cease to drive the blood and the patient will die It is probable that the inception of ventricular fibrillation is the cause of death in most patients who die suddenly from embolism or thrombosis of a coronary artery or of a large branch and also in many patients who have extreme dilatation of the ventricles due to cardiac failure Either of these conditions has been found in animal experiments to cause ventricular fibrillation and so can probably do so in human beings

Figure 61 shows the beginning of an attack of ventricular fibrillation in the human heart There are waves at the beginning of the attack in Figure 61 B which resemble deformed pre

the diaphragm and also from the variations which may result from the regular occurrence of an ectopic beat after each beat of the rhythm which is in control of the heart action The latter is

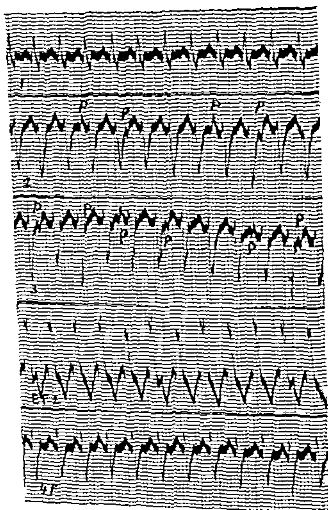


Fig 62 Nodal tachycardia with complete A V block showing electrical alternans of waves of the QRS group in all leads

particularly likely to be seen in attacks of paroxysmal tachycardia and has been called pseudoalternans by Wenckebach True electrical alternans has been observed only in the ventricular complexes of the human heart but it has been found in the P wave

described by Wiggers who has adequately discussed the theories of the mechanism of ventricular fibrillation

Ventricular fibrillation occurs experimentally in hearts which are subject to various sorts of poisoning In human hearts it may occur in lobar pneumonia or from the combined action of chloroform plus asphyxia It also occurs in severely failing hearts and after coronary artery occlusion Tying off a main coronary artery in a dog frequently leads to a period of ventricular tachycardia which passes into fibrillation of the ventricles

Schwartz and Jezer have demonstrated that it is a rather common cause of Adams Stokes attacks in patients with complete heart block and have also observed it in patients with normal rhythm and with partial A V block It may be suspected as the cause of syncopal or convulsive attacks in patients with complete heart block when the patient is observed to have frequent premature beats in addition to the basic idioventricular rhythm and especially if two or more of these beats occur together as in Figure 61 A The record of Figure 61 B shows the beginning of a long attack in another patient with complete heart block One of the rhythmic beats of complete heart block is followed by a premature beat The next rhythmic beat is aberrant and is followed by what resembles a deformed premature beat which begins the series of varying and somewhat irregular oscillations which indicate ventricular fibrillation and which continue through records C and D The character of the oscillations is characteristic resembling as it does a series of deformed ventricular ectopic beats their form continually varying from one complex to another The diagnosis of such attacks must obviously depend upon obtaining an electrocardiographic record but the finding of the preliminary phase of coupled beats and groups of two or more ectopic ventricular beats should strongly suggest that attacks of asystole are due to ventricular fibrillation

Electrical Alternans

The term electrical alternans is applied to the occurrence in the course of a regular rhythm of alternate beats with two different types of complex It must be distinguished from the variation in the electrical complexes which results from the movement of

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of records of the dying heart of the dog and in the auriculoventricular conduction time of the turtle. It is considered to be due to the failure of some particular myocardial area to respond to the contraction stimulus in alternate heart cycles.

Electrical alternans is not usually accompanied by the phenomenon of pulsus alternans although in certain instances they may occur together. Figure 62 shows a nodal tachycardia which probably originates above the branching of the A V bundle and is associated with partial right bundle branch block. This is indicated by the prolonged QRS duration of 0.12 second, the broad S wave in Lead 1 with deep S in Leads 2 and 3 and by the late appearance of the R peak in Lead IV 2. The R waves of Lead 1 show a regular alternation in height except for the eighth cycle whose R wave is equal in height to the ones preceding and following it. In Lead 2 the S wave shows a regular alternation in height except for the sixth and ninth cycles which also have peaks equal to those on either side. In Lead 3 there is an alternation of S which is regular throughout although in this lead as well as in Lead 2 the variation in height due to respiration makes the alternation more evident in some parts of the record than in others. There is alternation of the R wave in Lead IV 2 except in the first two cycles and of the S wave in Lead IV except for the fifth cycle. In such a record as this the electrical alternation may be due to alternating conduction in a portion of the affected bundle branch. A record has been published by Hamburger which illustrates alternation of the R wave with a regular normal sinus rhythm and normal ventricular complexes.

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[1] Some of the cases reported here are certainly not flutter but the illustrations are valuable particularly Figures 8 and 12

pulse until after a time the ventricles would become roused to an activity of their own (ventricular escape) and would begin to beat usually at a regular rate of about 30 beats per minute As far as is known auricular stoppage is always followed after an interval by spontaneous ventricular activity unless the auricles should begin first in which case the normal succession of the heart beat is again carried on

When the heart is irregular the size of each pulse wave is dependent upon the length of the pause preceding it Variations in the length of diastole will cause variations in the degree of filling of the ventricles If the pause is long the ventricles will be well filled and the pulse wave will be large if short there will be but little blood to be ejected into the aorta by the contraction and there may be only a small pulse or perhaps none at all Pulse rate or rhythm should never be taken to indicate heart rate or rhythm The physician should always listen over the apex of the heart to ascertain the regularity or irregularity of the heart action

The electrocardiogram has contributed greatly to the understanding of abnormal cardiac mechanisms so that now we are able to diagnose them correctly in the majority of instances without a record being taken There are many times however when a record will show our clinical diagnosis to be incorrect and there are certain conditions such as prolonged auriculo ventricular conduction time which would never be suspected without instrumental aid

Sinus Depression

Sinus arrhythmia is extremely common A slight degree of arrhythmia can be found on measuring the records of almost every heart whose rate is under 80 per minute This would be almost or quite imperceptible to one listening at the apex beat or feeling the radial pulse A degree of sinus arrhythmia which can be observed by these latter methods is also quite common especially in those under thirty and over fifty years of age and in any person after defervescence from an acute infection It is noted in practically everyone when the heart slows after it has been caused to accelerate by exercise

CHAPTER VII

CLINICAL ASPECTS OF DISTURBANCES IN RATE OR RHYTHM

SINCE we are concerned mainly with the various aspects of the electrocardiogram the title of this chapter takes us somewhat outside of the particular field It leads us into one which has been very well covered in other publications and so a complete discussion of the subject is not attempted This chapter will summarize those features in connection with the disturbances in rate or *rhythm which are considered of most clinical importance*

When the mechanism that produces the normal sinus rhythm is disturbed the ventricular systoles may be affected variously They may become irregular without change in rate or may become rapid and regular or in other instances slow and regular Occasionally even the rate may be regular and within normal limits with an abnormal mechanism in control We shall consider the clinical aspects of the abnormalities of cardiac rhythm together in this chapter even though the result may sometimes be a regular heart beat and at other times an irregular one The irregularity or lack of it is not so important as the fact that the *heart beat has an abnormal origin*

When considering disturbances of the rate and rhythm of the heart it is necessary to keep in mind the *relation of the auricular systole* to that of the *ventricles* and also because the pulse is so often carelessly taken to indicate the heart action the *relation of the ventricular systole* to the occurrence of a *pulse wave* Under normal conditions each auricular systole is followed by a ventricular systole and this is followed by a pulse wave which passes to the periphery If the auricles for some reason should omit one beat the ventricles would also omit this beat and there would be one pulse beat missing If the auricles should stop for a long period the ventricles would also stop and there would be no

beats from the normal auricles or ventricles by electrical stimulation of the sympathic nerves by the combined action of chloroform and asphyxia and by the use of such drugs as morphine strophanthine digitalis adrenalin and caffeine

The human heart also may give rise to physiological premature beats and these may arise in any part of the heart They are frequently found as a result of Graves disease and are probably often due to the overactivity of the autonomic nervous system which accompanies this condition They may possibly be due to the action upon the myocardium of the excessive thyroxin secretion They occur also in psychoneurotic individuals and probably then result from the generally exalted nervous activity

Premature beats commonly occur in association with organic disease of the gall bladder and less commonly with disease of the stomach intestines kidney or lungs The mechanism here is probably a reflex through the autonomic and sympathetic systems Mackenzie s interesting observation that frequently the hearts of normal women show premature beats at some time or other during pregnancy should be mentioned here The mechanism of their production is not known but it well might be a reflex from the abdominal viscera

Premature beats may result from the action of drugs in the human heart also digitalis morphine coffee tea and tobacco being of most clinical importance Stopping the drug will promptly abolish the arrhythmia if it should be the cause

Besides these not uncommon extracardiac causes of premature beats many patients with this irregularity have as a cause a pathological condition in the heart The diseased area need not be a large one and its seriousness for the heart as a whole is often slight A small focus of streptococcic or other infection or a small area of deficient blood supply due to coronary narrowing is capable of causing this disturbance and might not affect the heart s function appreciably

The importance of the premature beat is here seen to depend upon the importance of the causative disease

In a series of 127 cases with premature beats studied by Lewis 71 per cent of the patients were affected by one or another defi

This arrhythmia is usually symptomless but the more extreme types of sinus depression such as sudden temporary heart stoppage, or periods of very slow heart are likely to cause dizzy spells or palpitation. With the carotid sinus syndrome there may be fainting or even convulsions. No matter how severe these symptoms may be there often is no organic cardiac disease. The clinical significance of an arrhythmia due to a depression of sinus activity is usually that of an overactivity of the vagus nerve even though the arrhythmia may have caused symptoms referred to the heart or circulatory system. The disease if any is almost always extracardiac and the symptoms are due to a temporary functional depression of the heart rate and to cerebral anemia from the resulting decreased output of blood.

Occasionally arteriosclerotic changes will lead to a diminished blood supply to the sino-auricular node with depression of its function. Pressure due to tumor or infection in the neck may cause an increased vagal activity as may also increase intracranial pressure due to tumor or hemorrhage. An abnormally increased irritability of the carotid sinus may cause excessive vagal activity resulting in one or more of these manifestations of sinus node depression. Very rarely we may encounter a much slowed sinus rhythm possibly combined with a sinus arrhythmia which is due to a depression of the sinus node because of an acute inflammatory or chronic degenerative process within it. This must be recognized by its association with other signs of the presence of the causative heart disease. The prognosis and treatment of these arrhythmias depend more upon the etiology than upon the rhythm disturbance though the clinical manifestations may be due entirely to the slow heart action.

Premature Beats

The clinical significance of premature beats is very variable from one case to another for it depends partly upon their cause and partly upon whether their origin is in the auricles, the ventricles or the junctional tissues. A premature beat may always be viewed as an expression of a hyperirritable locus in the heart.

In experimental animals it is possible to produce premature

periments show no change worthy of note When the premature beats come more often they cause a considerable falling off in the blood flow

From quite another point of view a greater frequency of occurrence is an undesirable prognostic factor Frequently occurring premature beats especially if they occur irregularly usually are caused by a disease of the myocardium rather than by nervous influences while the more infrequent ones are usually due to nervous influences This statement is not without special exceptions in both of its phases especially when the premature beats occur regularly after every other normal beat These are often of a benign origin When two or three premature beats occur in succession the cause is almost always myocardial disease

The circumstances under which the premature beats occur also give a clue to their importance When they appear after digitalis administration they have no significance other than that the drug is causing what has been called one of its minor toxic phenomena When tobacco tea coffee or alcohol are used to excess the premature beats may be due to these drugs and may disappear when they are stopped

Premature beats sometimes are present only when the patient is quiet and the heart rate slow and disappear when the rate becomes more rapid from whatever cause In such cases the premature beats are especially likely to occur when the heart slows down just after a period of exercise probably because of the activity of the vagus and sympathetic system at this time These are often benign cases without a serious foundation

In other patients the premature beats will become more frequent when the heart is accelerated by exercise and these usually are due to a myocardial *focus* When premature beats occur in the course of an acute infection such as scarlet fever rheumatic fever or pneumonia they may be considered to indicate an invasion of the heart muscle by the disease

The prognosis quite evidently *does not* depend upon the presence of the premature beats so much as upon their cause We shall feel that they are due to heart disease if such other signs are present as the murmurs of valvular disease an abnormal electrocardiogram of the rhythmic beats a decrease of the cardiac

nite form of disease of the heart and the other 29 per cent could be divided as follows 19 per cent had active disease elsewhere in the body such as bronchitis lumbago gastric disease and so on (2 cases of exophthalmic goiter are included here) and 10 per cent, except for the irregularity were apparently healthy In a series of 50 cases which I have reviewed from this standpoint the proportions in these groups are 84 per cent with cardiac disease 10 per cent with active disease elsewhere and 6 per cent without any disease being found The difference between these two sets of figures is not significantly great As both Lewis and the author s series are taken from groups of individuals who predominantly have more or less serious cardiac disease it is only surprising that so many with the irregularity did not also have evident disease

It has been stated that when cardiac disease is not present the irregularity may be due to reflex or other nervous causes but it must be borne in mind that premature beats occasionally may be the only evidence of myocardial disease They may be due to the activity of a disease focus not large enough or not properly situated to give rise to any other sign When we consider how insidious the onset of arteriosclerotic heart disease must be we must realize that when this arrhythmia appears for the first time *in patients over forty years of age there is a definite possibility* that this may be the first indication of a small localized area of myocardial degeneration or ischemia due to coronary narrowing

The *frequency of occurrence* of the premature beats has a bearing on their prognosis A premature beat entails a certain amount of wasted effort on the part of the heart for it contracts upon a ventricle which because of the short preceding diastole is only poorly filled with blood There can be but a small output of blood from this beat at best and there is often none at all yet the cardiac contraction expends as much energy as if the ventricle were full When the premature beat occurs infrequently it does not amount to a serious loss of cardiac power but the more frequently it occurs the more of the heart s energy is wasted It has been shown that in the dog artificially produced premature beats occurring four to eight times per minute change the blood flow but little the greatest loss being 11 per cent and some ex

evidence of cardiac involvement appearing during the course of a rheumatic infection pneumonia diphtheria or other acute disease It is common in these infections for the disease of node or bundle to be part of a more or less wide spread myocarditis Such acute processes increase the gravity of the outlook but should the patient survive they may resolve to such an extent that the conduction time becomes normal again and the heart may show no sign of having been affected The *complete heart block* which is common clinically may be due to repetitions of these minor insults but usually is a chronic destructive process involving the node or bundle The cause is sometimes rheumatic rarely syphilitic but usually arteriosclerotic With this etiology we can scarcely hope for a recovery of the conducting function so that the condition of the patient as well as his outlook for the future will depend upon the integrity of his ventricular muscle how much it is affected by the disease and how well it is able to compensate by an increased strength of the individual beats for the decreased frequency of the contractions

In *partial block* the immediate outlook is governed but slightly by the number of dropped beats per minute though a rate under 50 per minute is a definite mechanical disadvantage The quality of the ventricular muscle its relative freedom from disease is always the predominant factor in the prognosis and for this reason the ventricular complexes should be examined for evidences of myocardial abnormality

TACHYCARDIA

There is a very distinct clinical separation between the tachycardia which we have termed physiological and that which is paroxysmal and might be called pathological The physiological tachycardia is the response of the heart to influences from the central nervous system or cardiac reflexes or to some toxin in the blood It occurs with fever hyperthyroidism excitement anemia cardiac failure and so on The rapid heart which occurs with intracranial conditions such as apoplexy is probably due to a depression of the vagus center releasing the heart from its normal retarding influences Physiological tachycardias do not necessarily mean disease of the heart and are never the prin

reserve power, sufficient cardiac enlargement to be unquestionable or an increase in the irregularity during the twenty seconds immediately after exercise If none of these things are observed we must conclude that the arrhythmia is not due to muscle disease of any extent or severity The next step is to look for the reflex or nervous factors which have been mentioned as possible causes of the arrhythmia If none of these are found we may feel that it is without clinical importance

It makes but little difference whether the disease focus is in the auricles or the ventricles The sequel of auricular myocarditis may be fibrillation of the auricles while that of ventricular myocarditis may be cardiac failure The sequel of myocarditis in the auriculoventricular node is heart block Disease is not usually sharply localized in any one situation for auricles node or ventricles are usually involved either in pairs or all together

The outlook of the patient with premature beats is plainly not affected by the irregularity It depends upon the cause of the irregularity the possibility of removing the cause and the effect upon the heart s efficiency if the causal condition should persist

Heart Block

The recognition of the higher grades of heart block is not usually difficult even without instrumental aid because of the slowness of the heart rate The rate is usually less than 10 per minute and the rhythm regular The lesser grades of block with dropped beats may be confused with premature contractions The pulse may be bigeminal or trigeminal just as when premature beats occur regularly One can usually distinguish which mechanism is present even without a record by listening carefully at the apex of the heart for the sounds of a premature beat Pressure on the carotid sinus will often help our decision for it will increase the frequency of dropped beats due to heart block though it will not increase the frequency of premature beats

The lowest grade of block with *prolonged conduction* time between auricles and ventricles cannot be determined without the polygraph or the electrocardiograph Prolonged conduction time perhaps with occasional dropped beats may be the only

so that the ventricles are slower and perhaps irregular In the latter case the heart beat simulates very closely indeed the rhythm of auricular fibrillation The reaction to carotid sinus pressure by a slowed and irregular ventricular response is a characteristic feature of flutter but this change lasts only during the pressure and the rapid regular rate promptly returns Digitalis administration will usually slow the ventricular rate when the patient s therapeutic dosage is reached and all patients with this condition should have the rate properly controlled by digitalis medication Under digitalis the heart usually becomes irregular as well as slower

Auricular flutter is usually dependent upon a pathological process although it may be toxic in origin and perhaps very rarely may be neurogenic Careful microscopic studies of the auricular muscle in this condition are not numerous but they usually show a diffuse fibrosis perhaps due to chronic arterial disease or to a diffuse leucocytic infiltration It occurs especially in chronic arteriosclerotic patients and in others who have an interstitial myocarditis whether due to acute rheumatic fever lobar pneumonia diphtheria or after syphilitic infection A patient having an attack during an acute infection may recover from it and subsequently have no signs of cardiac abnormality On the other hand those who have auricular flutter as the result of chronic processes often have some limitation of their cardiac reserve in the intervals between attacks This shows that the ventricles as well as the auricles are affected by the myocardial disease in these cases

Patients who have had one attack unless it occurred during an acute infection are very likely to have another They are also likely to have auricular fibrillation either coming on suddenly with an attack of rapid irregular ventricular action or as a sequel to a period of flutter The physiological relation between flutter and fibrillation of the auricles appears to be extremely close certain cases changing repeatedly from one mechanism to the other

The rapid ventricular action due to auricular flutter can usually be readily controlled by digitalis This drug also affects the auricular activity frequently changing flutter into fibrilla

cipal cause of heart failure though they may be a contributing cause by leading to cardiac fatigue Long continued rapid heart action will lead to undernourishment of the heart muscle and consequently to cardiac fatigue and failure but usually the toxemia which causes the rapid rate damages these hearts more than the rate itself

Paroxysmal tachycardia is often due to disease and often not This is of course a distinction of importance and is to be determined along such general lines as have been detailed for premature beats (page 211) The clinical significance of paroxysmal tachycardia differs according to the part of the heart in which it originates Paroxysms arising in the auricles and in the auriculo ventricular node are more often due to a disturbance of the cardiac nervous mechanism than to disease Especially in young people, they often cease to occur after a time and the heart seems to remain intact

Ventricular tachycardia rarely if ever appears except as a result of cardiac disease It is especially liable to be caused by disease of the coronary arteries

The clinical importance depends greatly upon the readiness with which the attacks can be stopped Auricular and nodal foci are more under the control of the vagus nerve than are ventricular foci and carotid sinus pressure or other forms of vagus stimulation including drugs which have this action are more likely to stop attacks originating from these foci than those originating in the ventricles The prognosis of tachycardia of auricular or nodal origin is therefore better in this respect also

Auricular Flutter

Auricular flutter may come on in sudden attacks as does paroxysmal tachycardia and these attacks may end as suddenly Without an electrocardiogram it cannot be diagnosed certainly except after several observations Auricular flutter will sometimes occur in a chronic form persisting for months or years or it may give place to auricular fibrillation Since the auricular rate is usually between 260 and 320 per minute the usual 2 1 A V ratio makes a ventricular rate close to 150 the usual finding In long continued cases the A V blocking is usually greater

reversion to normal rhythm Thus for many patients the onset of permanent fibrillation can be considerably postponed

When fibrillation of the auricles is permanently established the prognosis for each individual case demands above all else that the proper amount of digitalis be used to slow the ventricular rate and keep it continually between 70 and 80 per minute while the patient is at rest Without such medication the tendency is almost universally to heart failure through exhaustion of the heart muscle by the rapid ventricular beating With occasional patients who have a pathological lesion in the A V system or a vagotonia so that a certain amount of depression of A V conduction is present the rate does not tend to be rapid even without digitalis

The prognosis depends first of all upon maintaining an approximately normal ventricular rate using digitalis if this is necessary Even the best heart will fail without this The prognosis when this is done depends as in all the other arrhythmias upon the degree of involvement of the ventricular muscle by the disease which has caused the arrhythmia and upon the mechanical handicap such as valvular lesions or high blood pressure against which the muscle must work

Auricular fibrillation is usually a result of myocarditis within the auricles It may be caused experimentally by the injection of certain drugs which damage the heart muscle and clinically may occur from acute toxic degeneration due to disease or to infection within the heart itself It often occurs as a result of overactivity of the thyroid Here it is usually in the paroxysmal form although occasionally it may be chronic The temporary development of auricular fibrillation has been reported as a feature of poisoning by hydrogen sulphide and also by carbon monoxide Very rarely it appears to be neurogenic especially under the influence of fatigue or excitement

The chronic form is usually due to a chronic fibrotic process within the auricular muscle This may be residual from a former acute condition or may be due to arteriosclerosis Rheumatic disease of the mitral valve is frequently associated with auricular fibrillation somewhat less than half of all cases showing evidence of mitral stenosis while about the same percentage are due to

tion If digitalis now be stopped the normal rhythm will some times return but fibrillation may continue or may revert again to flutter, so that we may be continually forced to deal with an abnormal cardiac mechanism Quinidine given directly the condition is recognized is usually though not always successful in causing a reversion to normal rhythm

If the ventricular rate is properly controlled by digitalis the patient can go about and do a great deal while auricular flutter is present The situation in this respect is quite similar to that with auricular fibrillation except that the ventricular rate is somewhat more difficult to control in the presence of flutter The ultimate outlook for these patients depends more upon the degree of valvular disease or hypertension the integrity of the ventricular muscle and the proper control of the ventricular rate by digitalis than upon the character of the auricular activity The prognosis for the attack with the use of proper treatment is good but the ultimate outlook is to be determined by the extent of the cardiac disease on such grounds as are discussed under auricular fibrillation

Auricular Fibrillation

Clinically auricular fibrillation appears in a paroxysmal and also in a chronic form The chronic cases often give a history of having had one or more paroxysmal attacks before the onset of the one which finally persists As has been mentioned auricular fibrillation may be a sequel to auricular flutter When this occurs the fibrillation may either revert to flutter give place to normal rhythm or remain persistent

The paroxysmal form of auricular fibrillation as it appears clinically differs from paroxysmal attacks of auricular flutter only in the irregularity of the ventricles The symptoms treatment and prognosis are practically the same When auricular fibrillation is long established it is usually permanent though even long standing cases have been observed to revert for a time to normal rhythm Quinidine when properly given frequently will cause even long standing auricular fibrillation to give place to normal rhythm and this will often remain for a considerable period If the fibrillation returns quinidine may again cause

CHAPTER VIII

VARIATIONS OF THE ELECTROCARDIOGRAM RESULTING FROM DISEASE AND OTHER ABNORMAL INFLUENCES

IN PREVIOUS chapters the characteristics of the normal electrocardiogram have been reviewed and those electrocardiographic abnormalities which may result from disease or abnormal function of the heart have been described We shall here consider what sort of information the records are able to give in what types of patients the records are likely to be of diagnostic importance and what particular electrocardiographic abnormalities are likely to be found as a result of the different diseases and intoxications that affect the heart This chapter is written to point out the limitations of this method of examining the heart and to indicate when it will be found especially valuable

How to Read an Electrocardiographic Record

When examining an electrocardiographic record it is necessary to have a definite method of procedure in order to avoid missing important but unobtrusive features such as for example a slight prolongation of the P R interval or of the QRS group Having a definite order for examining the numerous details of the record nothing important will escape notice The following order is suggested as being logical and complete

1 Examination of the test of standardization to determine

a Whether the jump is exactly 10 mm

b Whether there is overshooting at the end of the jump

2 Determine the P wave in each lead

3 Determine the rate of the auricular systole The number of complete heart cycles (P P interval) in 6 seconds (30 of the fifth second divisions usually occupying about 15 cm of the

arteriosclerotic myocardial conditions There are however a certain number of hearts with auricular fibrillation whose auricles reveal under the microscope comparatively little disease Probably in these and in all paroxysmal cases abnormal nervous impulses are an important factor in precipitating the onset and the fibrillation once started tends to perpetuate itself Cases with slight pathology are possibly those that revert spontaneously to normal rhythm or do so easily under quinidine Cases that do not revert under quinidine are possibly those with more marked structural changes in the auricular muscle

A pathological process that involves the auricles may involve the ventricles but little This is the reason why some hearts with auricular fibrillation are able to carry on the circulation so well even under the handicap of valvular disease high blood pressure or continual strenuous bodily exertion provided that the rate is kept from becoming too rapid for the ventricles to function at their best If the ventricular complexes are of normal form and the valvular disease or increased blood pressure not too extreme the patient s cardiac reserve will often be good enough to allow him to do what he wishes with but slight limitation If these other factors are unfavorable the resulting handicap will probably cause a certain amount of crippling of the patient s ability The actual irregularity itself provided the rate is kept within normal limits by proper treatment probably handicaps the heart but little

18 Note the R wave its height with the different positions of the electrode the time of its apex after the beginning of QRS with the different positions of the electrode

19 If Q is present measure its height Is it normal?

20 If S is present measure its height Is it normal?

21 Measure the level of the S-T junction Is this normal?

22 Is the T wave upright or inverted

23 Note the form of the S T segment

24 Measure the height of T

Information Derived from Records

Like any other single method the electrocardiographic examination has more value for some purposes than for others It can

1 Tell whether the heart beat is governed by stimuli from the normal site

2 Indicate the mechanism of any arrhythmia that may be present

3 Give a sign of auricular hypertrophy

4 Record the auriculoventricular conduction time

5 Suggest in the presence of cardiac enlargement that the right or left ventricle is preponderantly hypertrophied or that neither is preponderant

6 Give an indication of the physiological condition of the ventricular muscle

7 Give evidence of the action of certain drugs upon the heart (digitalis quinidine nicotine etc)

8 Give evidence of the action of certain toxins upon the heart (thyroid) or of physiological disturbances of the metabolism of the myocardium (beriberi)

9 Show certain peculiar features of the waves which have resulted from disease of the ventricular muscle

The electrocardiogram gives its own special variety of information namely the manner of development of the electrical potential during the cardiac contraction From the curve we may draw deductions concerning the mechanism and character of the contraction of the muscle fibers of the heart An examination of the heart is as incomplete without the electrocardiogram as

record) is to be multiplied by 10 and a figure added to represent any fractional part of a heart cycle which may have been included within the 6 second interval

4 Does P occur regularly or irregularly? If irregularly have the P waves after the short pauses the same form as the others (sinus arrhythmia) or a different form (premature beats)?

5 Have the P waves constantly an abnormal form (height inverted notched wide)?

6 Are the waves of auricular flutter or auricular fibrillation present instead of normal P waves?

7 Measure the P R interval in order to appraise the function of auriculoventricular conduction Does the P R interval vary? Is the ventricular complex missing after certain P waves?

8 If heart block auricular flutter or auricular fibrillation is present determine the rate of ventricular systole by counting the number of P R intervals in 6 seconds as described above

9 Does the ventricular complex occasionally occur after a *short P R interval? If so has this ventricular complex* the appearance of the usual complexes of this record indicating a nodal premature beat or a different appearance indicating a ventricular premature beat?

10 Are the ventricular waves the same throughout each lead? If not determine the cause for the variation

11 Does the QRS group show either right or left axis deviation?

12 Does QRS constantly show (a) notching or slurring (b) abnormal voltage (c) abnormal duration?

13 Does the S T junction occur at or sufficiently near the isoelectric level to be normal or is it abnormally elevated or depressed? If so note the relation of the elevation and depression in the three leads

14 Note the character of the curve from the S T junction to the peak of T

15 Does T constantly show an abnormal voltage an abnormal direction or an abnormal form?

In the precordial leads

16 Observe the P wave and measure the P R interval

17 Note the QRS duration

cases these complicating conditions are not present and the unusual axis deviation cannot be explained. Though this is relatively infrequent, yet the possibility must be emphasized in order that the table may not mislead by its diagrammatic appearance.

TABLE IX

EFFECT OF VALVE LESIONS UPON ELECTRICAL AXIS OF QRS

Valve Lesion	Usual	Unusual	Coexisting Conditions Which Would Explain the Unusual Direction of the Axis of QRS in the Presence of the Valve Lesion in Question
Mitral insufficiency	Slight left axis deviation	1 Normal direction of axis of QRS	1 Early mitral stenosis 2 Marked pulmonary emphysema or chronic tuberculosis 3 Long narrow chest with vertical heart
		2 Right axis deviation	1 Mitral stenosis 2 Combination of two or more of above factors
Mitral stenosis	Right axis deviation	1 Normal direction of axis of QRS	1 Early slight stenosis with long standing insufficiency 2 High blood pressure 3 High diaphragm with transverse heart 4 Aortic insufficiency
		2 Left axis deviation	1 High blood pressure 2 Aortic insufficiency 3 Combination of two or more of above factors
Aortic insufficiency	Left axis deviation	1 Normal direction of axis of QRS	1 Mitral stenosis 2 Marked pulmonary emphysema or chronic tuberculosis 3 Long narrow chest with vertical heart
		2 Right axis deviation	1 Mitral stenosis 2 Combination of two or more of above factors

Combined lesions are a more complicated problem, but when the heart is enlarged and abnormal duration or marked notching of QRS is not present to indicate an abnormal path of the excitation, its electrical axis will indicate the ventricle showing preponderant hypertrophy with a fair degree of accuracy. If the direction of the axis of QRS does not agree with the hyper

it is without auscultation *or a determination of the size of the* heart By omitting any one of these methods we may deprive ourselves of information of considerable value in the diagnosis

Because of the effort necessary to obtain an electrocardiographic record this method of examination must be omitted more often than simpler methods For this very reason the clinician should have clearly in mind the kind of information to be obtained from it He should know in what types of cases the information of the electrocardiogram is imperative and also what is the likelihood when he omits this examination that he is missing an important feature of his patient s disease

The problems which the physician should refer to the electrocardiograph for solution will be perhaps more easily appreciated if presented as clinical entities Therefore various types of patients with heart disease will be considered and the importance of the electrocardiographic information will be discussed in each category To group the patients for this presentation prominent clinical features will be used such as the presence or absence of symptoms referable to the heart (dyspnea edema or pain on effort) valvular disease cardiac enlargement and irregular heart action as well as the influence of certain pathological entities such as congenital abnormality or coronary thrombosis or of certain etiological factors such as rheumatic fever or diphtheria

Valvular Disease with Regular Heart

Patients with valvular disease and a regular heart with or without cardiac enlargement frequently have an electrocardiogram indicating preponderant hypertrophy of one or the other ventricle This finding may be of assistance in the diagnosis of the variety of valvular disease provided that the evidence from the murmurs alone is not conclusive The subject has been thoroughly discussed in Chapter III and Table IX may serve as a summary of what has already been said It must be emphasized that this is a diagrammatic presentation and that there are not infrequent exceptions to the usual findings here indicated In many cases these exceptions may be explained by the presence of the conditions listed in column I of the table but in other

Without Valvular Disease or Arrhythmia

Patients with symptoms referable to the heart but without valvular disease or arrhythmia may be suffering from neurocirculatory asthenia or hyperthyroidism or coronary arteriosclerosis Those with neurocirculatory asthenia do not usually show abnormalities of the ventricular waves for there is no myocardial disease present in these cases but occasional exceptions are found as will be mentioned later when the electrocardiographic findings in this condition are discussed in detail The effects of hyperthyroidism and of coronary arteriosclerosis upon the electrocardiogram are discussed in detail later It will suffice to say here that those with hyperthyroidism rarely show abnormalities of the electrocardiogram to indicate myocardial disease unless the condition is marked while those with coronary arteriosclerosis often do so even though the heart may not be enlarged

Patients in this group who have cardiac enlargement and symptoms of circulatory insufficiency often show abnormal electrocardiograms They may have a diffuse myocardial degeneration due to coronary narrowing or to multiple foci of inflammation within the muscle and abnormal ventricular waves are found in about 85 per cent

Congenital Abnormalities

Patients with congenital abnormalities of the heart often give an electrocardiogram with right axis deviation of QRS and ...

... ventricle and the resulting hypertrophy causes right axis deviation of QRS Neither patent foramen ovale nor intraventricular band usually leads to changes in the electrocardiogram Hearts with congenital abnormalities often have variations in the distribution of the auriculoventricular conduction system These variations occasionally cause abnormal duration and notching of QRS as is seen in Figure 39

Recently Schnitker has collected the electrocardiograms of over 100 autopsied cases and grouped them according to the predominant congenital defect The most important congenital

trophy that might be expected to result from the influence of the conditions we have diagnosed we should then carefully seek for the reason of the disagreement Perhaps if right axis deviation is present in association with aortic insufficiency we may be led to change our interpretation of a diastolic rumble at the apex and consider it due to mitral stenosis rather than to the Flint mechanism Perhaps in a search to explain a left axis deviation we may attach a greater significance to a slightly raised blood pressure or find a faint murmur of aortic insufficiency which had previously been missed

The direction of the electrical axis of QRS is influenced by other factors than those shown in the table and occasionally an explanation of the findings *in a given case* will be impossible *This may be because of influences of which we are unaware in* the particular case or because we are unable to properly evaluate the resultant of several influences which may be *present*

Irregular Heart Without Valvular Disease

In patients with irregular heart action without valvular disease and without cardiac enlargement or symptoms of cardiac insufficiency the arrhythmia will almost always be due to premature beats It is rare for the electrocardiograms of these patients to show an abnormality indicating ventricular myocardial changes Perhaps in only 2 or 3 per cent will this occur and these will usually be cases with an arteriosclerotic etiology

When cardiac enlargement or symptoms of cardiac insufficiency or both are present *the irregularity is usually due to auricular fibrillation The record will generally show only a* left axis deviation of QRS of greater or less degree but perhaps 15 per cent will also have abnormal ventricular complexes indicating muscle disease This will usually be due to an arteriosclerotic form of heart disease though occasionally the etiology will be rheumatism or thyroid dysfunction Heart block occasionally occurs in patients without valvular disease but it is more usually associated with a slow and regular rhythm than with an irregular one that is block is more usually complete than incomplete in these cases

latter conditions will not cause the typical abnormal features in the record

Arteriosclerotic Heart Disease

Coronary Arteriosclerosis

The three main clinical manifestations of coronary arteriosclerosis are associated with abnormal ventricular waves with different degrees of frequency Patients who have *arrhythmia* from this cause whether auricular fibrillation auricular flutter heart block or premature beats are more likely than not to show ventricular waves which are normal except perhaps for a left axis deviation of the QRS group In these patients the disease is more marked in the auricles or in the junctional tissues or if the premature beats have a ventricular origin only a small ventricular focus need be affected In these cases the ventricles are often but little involved On the other hand a certain percentage of these patients will have besides the arrhythmia significant abnormalities of the ventricular waves such as described in Chapter ix

Patients who show *cardiac insufficiency* as the chief clinical manifestation usually are found to have abnormalities of the ventricular complex A considerable percentage of these patients will show abnormal inversion of T or notching of QRS or both Bundle branch block is not infrequent in these patients

Those who show the anginal syndrome as their chief manifestation also are very liable to have abnormalities of the ventricular waves Of the patients who suffer from the angina of effort about one third will show the coronary T wave Another third will show other abnormalities of QRS or of T such as notching or slurring of QRS or inversion of the T wave a large Q wave in Lead 3 is not uncommon in records from this type of patient The remaining third of this group will not show any significant abnormality of the ventricular complex except perhaps a left axis deviation of QRS In such patients it is likely that the coronary arteriosclerosis has not yet progressed sufficiently or that the resulting coronary insufficiency has not been present long enough to have given rise to pathological changes in the myocardium It must be borne in mind though that minor myocardial changes may fail to give rise to electrocardiographic abnormali

lesions were associated with special electrocardiographic features as follows:

Coarctation of aorta with left axis deviation of QRS.

Patent foramen ovale with no special features. Even if a large opening is present there is only a slight tendency to have right axis deviation of QRS though large P waves and auricular fibrillation also occur.

Interventricular septal defects with *negative or slight right axis* deviation of QRS and possibly evidences of disturbed A-V conduction or bundle branch function.

Patent ductus arteriosus with no abnormal axis deviation of QRS.

Pulmonic stenosis with marked right axis deviation of QRS and large P waves particularly in Lead 2.

Tetralogy of Fallot with moderate to marked right axis deviation of QRS, *possibly also increased* A-V conduction or intraventricular conduction.

Congenital *transposition* of the heart, its apex being turned toward the right instead of the left, gives rise to an electrocardiogram suggesting right axis deviation by the small or absent R *and deep S in Lead 1, but can be distinguished from this by the* fact that not only is the predominant wave of the QRS group directed downward in Lead 1, but the P and T waves also are downward in that lead. It is as if a normal Lead 1 were turned upside down, and this is practically what it is, for the right arm wire, which is normally *nearer to the auricles* and the basal part of the ventricles, is now, because of the transposition of the heart, nearer to the apical part of the ventricles. Likewise the left arm wire, *instead of being nearer to the apex, is now nearer to the* base of the heart. The currents in the heart develop normally but, owing to this different leading off from the heart to the galvanometer, the curve by Lead 1 is turned upside down, that by Lead 2 is what we ordinarily obtain by Lead 3 and that by *Lead 3 is the ordinary* Lead 2 *considering their true relation to* the heart. A reference to the diagram of Figure 2 will make this plain. The typical electrocardiogram described will aid in distinguishing a congenital transposed heart from one which lies on the right side, because of pleural or mediastinal disease. The

more of the limb leads it is found to be situated definitely above or below the zero level In certain records the deviation may be so slight that it does not exceed that shown by excep-

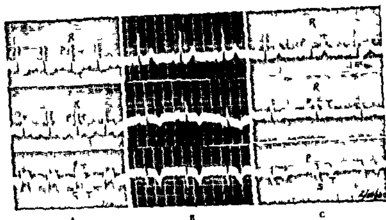


Fig 63 Three records from a patient showing the progressive changes after cardiac infarction affecting the diaphragmatic portion of the left ventricle The changes are of the Q 3 T 3 type

A This record was made the day following the attack and shows an elevation of the S T junction in Lead 3 while in Lead 1 this portion of the curve is below the zero level

B This record obtained seventeen days later shows the change of T 2 and T 3 to inverted waves suggesting the coronary feature but not showing it typically T 1 now has a normal appearance The QRS group of Lead 3 is now represented by a QS wave (marked S)

C This record obtained two years after B shows a T wave which viewed as an isolated feature could not be called abnormal although it is downward in Lead 3 and has only a small amplitude in Lead 2 The QS wave (marked S) still persists

tional normals (Fig 14) but typically it exceeds the limit of normal variation by from 1 mm to 5 mm in different leads From the S T junction the curve passes to the peak of T by a straight line or by a curve that is concave toward the baseline so that if it arises above the baseline the peak of *T* frequently shows a broad rounded contour as in Lead 3 of Figure 63 A in Lead 1 of Figure 64 A and in Leads 2 and 3 of Figure 65 A Because of the breadth of the peak of the T wave the descending limb is quite abrupt as in Lead 3 of Figures 63 A and 65 A (See also Fig 1* C and D)

This deviation of the S-T junction and the broad rounded or plateau like T wave is associated with a localized area of acute

ties Occasionally patients with a normal electrocardiogram who may or may not have had preceding anginal symptoms die suddenly from coronary thrombosis This shows clearly that an artery may be seriously diseased and yet not cause recognizable change in the ventricular complex

Records made during attacks of angina pectoris have frequently been observed to show changes in the S T segment and T wave which disappear after the cessation of the attack These changes usually consist of a departure of the S T junction from its previous level and sometimes also a variation in the appearance of the S T segment so that the record may slightly suggest the appearance characteristic of infarction or more closely resemble the changes found during healing of an infarct Coincident with these changes or even without them the T wave may become smaller or may become inverted In occasional instances a bundle branch block curve was present during an attack of angina pectoris but was not present in the intervals between attacks

MYOCARDIAL INFARCTION

Patients with myocardial infarction due to coronary thrombosis will show an abnormality of the electrocardiogram in practically every case if more than one record can be obtained during the first week A single record will occasionally fail to show diagnostic changes for these are sometimes late in their onset and sometimes transient As a rule however changes are present within a few hours and persist for several weeks or even months Because the changes occasionally are transient records taken after the second week or even earlier in some cases may fail to show evidence of an abnormality which was previously present An infarction which arises in the course of extensive coronary arteriosclerosis may not be due to a recent coronary thrombosis but the changes in the electrocardiogram are of a similar character

S T junction and T wave Soon after the occlusion the part of the curve most strikingly affected is the S T segment though QRS and T may also be changed (Figs 63 64 and 65) The typical feature of this stage is that the S T junction does not lie as normally near the baseline of the record but in two or

has been described as the coronary T wave is produced (Lead I of Fig 64 c and of Fig 68 B) This consists of an upward convexity of the S T segment the remnant of the original broad

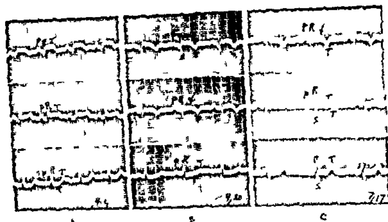


Fig 64 Three records from a patient showing progressive changes following cardiac infarction affecting the anterior part of the left ventricle The changes are of the Q I T I type

A This record obtained two hours after the attack began shows an elevation of the S T junction in Leads 1 and 2 and a depression of this point below zero in Lead 3 There is low voltage of QRS and a small Q 1

B This record obtained sixteen days later shows a change of T 1 and T 2 to typical coronary T waves d phasic (+ −) waves with an upward convexity of the first limb of T T 3 still shows some depression of the S T segment

C This record obtained three months after record B shows further changes in T 1 the S T segment now starting slightly below the zero level The T wave in Lead 2 is of very small amplitude and is diphasic (+ −) T 3 has a normal appearance The QRS voltage is low and leads 2 and 3 show R and S of about equal size

upward T followed by a downward T wave W[illegible]

[illegible] ...ie case may

...ws a gradual return of the S T segment to normal so that when the stage of the coronary T wave has been reached in the lead showing the major disturbance of T it is usual for the T wave in the reciprocal lead to have a normal appearance (Figs 64 C and 66 A) This series of changes was first demonstrated in the human electrocardiogram by the author in a case showing the typical features in Leads 2 and 3 and has subsequently been confirmed by Parkinson and Bedford and later observers Smith

degeneration of the ventricular myocardium such as is produced by infarction It was first observed by Eppinger and Rothberger following the injection of silver nitrate and other coagulative poisons into the ventricular musculature and first in clinical records by the author Parkinson and Bedford were first to point out a reciprocal relationship of the S T segment and T wave changes in Leads 1 and 3 When the typical elevated S T junction *is found in one of these leads there will be a depression of the* S T junction in the other lead The subsequent course of the T wave in this reciprocal lead is occasionally symmetrically similar to that of the curve following the elevated S T junction in the typical lead except that the wave is inverted Usually, however the depression is not so marked in the reciprocal lead as is the elevation in the typical lead and it is usual to find that *following the depressed S T junction the S T segment crosses the* zero level to form a small upward T wave as seen in Lead 1 of Figure 65 A and Lead 3 of Figure 64 A This wave is the reverse of that in Figure 42 B

The typical S T deviation of the acute phase may appear within *an hour or two of the time when the infarction occurs or may be* delayed until the third day or even later It may persist in typical form for a period which varies in different cases from twenty four hours to as much as ten or twelve days usually from five to ten days These variations probably depend upon the extent and situation of the lesion and the ability of the collateral anastomotic circulation to restore the injured area If marked *S T displacement persists for an unusually long time it is probably* due to a continued state of degeneration in an area of muscle or to a progressive degeneration of new muscle fibers

As *healing of the infarct* progresses a characteristic series of changes develops in the T wave of infarction The descending *limb of T begins to pass slightly below the zero level so that an* inverted peak follows the broad upward deflection which still remains This appearance is seen in Figure 42 B and in Leads 2 and 3 of Figure 63 A Lead 1 of Figure 68 A and to a very slight extent in Lead 3 of Figure 63 A The S T junction gradually approaches the zero level and as it does so *the downward peak* becomes deeper until eventually the diphasic (+—) form which

The typical S T deviation of the acute stage thus gives place to the coronary T wave in a variable time While this latter peculiarity may occasionally be established as soon as twenty four

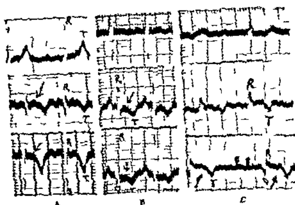


Fig 66 A and B Showing the upward convexity of the S T segment that appears with healing after coronary artery occlusion This curve is indicated by the arrows (the curve in Lead 3 of record A is not typical)

C Illustrating the stage of healing in the case from which 66 A had been obtained Note the downward T wave with elevated S T in Lead 3 and the depressed S T in Lead 1

hours after the attack it more usually appears between the fifth and the tenth day and sometimes not until twelve or fourteen days have elapsed Figure 63 A is a record obtained very soon after the attack Figure 63 C was obtained from the same patient four days later and Figure 66 A about two weeks after the attack Figure 63 B was obtained from another patient soon after the attack and Figure 66 C from this patient eight days later

Several authors have published electrocardiograms obtained from the human heart after ligating the anterior descending branch of the left coronary artery in the course of the surgical procedures resulting from a wound of the heart These curves closely resemble the ones described and clearly prove that these may be caused by coronary occlusion Similar changes followed the suturing of a wound in the anterior and apical part of the left ventricle even without the artery or vein being ligated so that it is evident that the changes in the S T segment and T wave are due to an area of degeneration in the myocardium

The displacement of the S T segment resulting from acute myocardial degeneration is probably due to the disappearance

had previously demonstrated it in dogs after ligation of a coronary branch and in association with Herrick had published a human record showing the coronary T wave in Lead 1 Parkinson

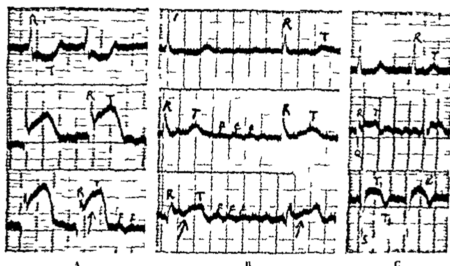


Fig 65 A Obtained soon after the occlusion occurred from the patient who later gave Figure 65 C and Figure 66 A The arrow indicates the elevated S T junction and broad rounded T wave which is the characteristic feature of recent infarction This is also present in Lead 2 while in Lead 1 the S T junction shows the typical reciprocal depression with broad rounded inverted S T segment (marked T) followed by a small upward T wave

B Obtained soon after the attack from the patient who later gave Figure 66 C Note the elevated S T junction and broad T wave in Lead 3 and slight depression of the S T segment in Lead 1 with upward T

C Intermediate between Figure 65 A and Figure 66 A to show the changes in the S T segment and the T wave which take place as the infarct heals The elevation of the S T segment in Lead 3 has become less and the latter part of T 3 has become a downward peak so that the wave is diphasic (+ −) and of the coronary type This is less evident in Lead 2 The small Q 3 of Figure 65 A has given place to a wide QS 3 (marked S) In Lead 2 however both Q and R are present

and Bedford were the first to point out that this series of changes in the S T segment and the T wave during healing would be observed in whichever lead showed the major disturbance of the S T segment with elevation directly after the infarction while the reciprocal changes would appear in the other No matter whether Lead 1 or Lead 3 shows the elevated S T junction Lead 2 usually shows changes of the general nature of those found in the lead with elevated S T segment but shows these changes to a less marked degree

The typical S-T deviation of the acute stage thus gives place to the coronary T wave in a variable time While this latter peculiarity may occasionally be established as soon as twenty four



Fig 66 A and B Showing the upward convexity of the S T segment that appears with healing after coronary artery occlusion The curve is indicated by the arrows (the curve in Lead 3 of record A is not typical)

C Illustrating the stage of healing in the case from which 6 A had been obtained Note the downward T wave with elevated S T in Lead 3 and the depressed S T in Lead 1

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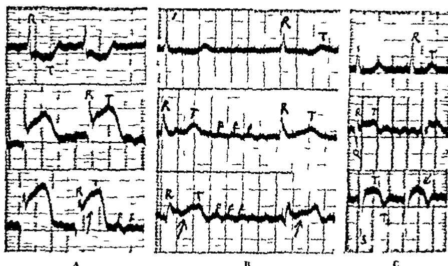


Fig 65 A Obtained soon after the occlusion occurred from the patient who later gave Figure 65 c and Figure 66 A The arrow indicates the elevated S T junction and broad rounded T wave which is the characteristic feature of recent infarction This is also present in Lead 2 while in Lead 1 the S T junction shows the typical reciprocal depression with broad rounded inverted S T segment (marked T) followed by a small upward T wave

B Obtained soon after the attack from the patient who later gave Figure 66 c Note the elevated S T junction and broad T wave in Lead 3 and slight depression of the S T segment in Lead 1 with upward T

C Intermediate between Figure 65 A and Figure 66 A to show the changes in the S T segment and the T wave which take place as the infarct heals The elevation of the S T segment in Lead 3 has become less and the latter part of T 3 has become a downward peak so that the wave is diphasic (+ −) and of the coronary type This is less evident in Lead 2 The small Q 3 of Figure 65 A has given place to a wide QS 3 (marked S) In Lead 2 however both Q and R are present

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The typical S T deviation of the acute stage thus gives place to the coronary T wave in a variable time While this latter peculiarity may occasionally be established as soon as twenty four

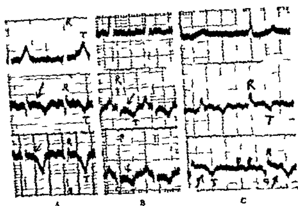


Fig 66 A and B Showing the upward convexity of the S T segment that appears curve is indicated by the arrows

ase from which 65 A had been evated S T in Lead 3 and the

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The displacement of the S T segment resulting from acute myocardial degeneration is probably due to the d[illegible]

during systole of the current of injury at the boundaries of the infarcted area The later T wave changes are probably the result of a prolongation of the duration of the contraction in the injured myocardial area The QRS changes later to be described and which are more permanent are probably due to the absence of the electric forces that were normally produced by the infarcted muscle

Changes in the QRS group Wilson and his associates have pointed out that records showing the elevated S T segment and later the coronary T wave in Lead 1 frequently also show a definite conspicuous and rather broad Q wave in this lead and a deep S wave in Leads 2 and 3 The QRS group of these records has its smallest deflections in Lead 1 (Figs 64 and 67) and often as in Figure 64 there is a low voltage of QRS manifested by low amplitude in all leads They also re emphasized the author's previous observation that records showing the elevated S T segment and later the coronary T wave in Lead 3 frequently showed a large Q wave in this lead They pointed out that some times there is no R wave in this lead as in Figures 63 B and C and 65 C so that the QRS group consists solely of a wide down ward deflection QS which they called Q because they believed its significance was the same as that of Q In records with large Q_3 or with QS_3 there is frequently also a Q wave in Lead 2 This lead usually shows the smallest QRS deflections of any of the limb leads (Fig 65 C)

They suggested that one might speak of a Q_1T_1 type of ventricular complex meaning that a prominent Q or QS wave and the elevated S T junction with the subsequent changes dur ing healing which have been described were found associated in Lead 1 One might also speak of a Q_3T_3 type of ventricular com plex meaning that analogous associated changes of QRS and of T were found in Lead 3 These typical associations are not infre quent but there are many records that fail to show a combination of QRS and T changes exactly fulfilling either category The T_1 changes are fairly common without the Q_1 feature and the QRS changes may be present in either lead with normal T waves The changes in the QRS group resulting from coronary narrowing or

occlusion are more permanent than the changes in the T wave so that a large Q_3 or Q_1 or the above described QRS complex with small deflections in Lead 1 and relatively large S and S_3 may

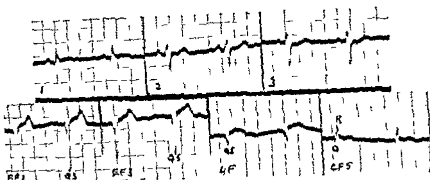


Fig 6 A record of the Q 1 T 1 type indicating anterior infarction Note the S T elevation in Lead 1 and depression in Lead 3 also the small R 1 with large S 2 and S 3 In the precordial leads note the absent P wave except beyond the apex and the elevated S T segment most marked at the apex and beyond

persist from an old lesion and complicate the picture of a more recent one

Barnes and Whitten first demonstrated that records corresponding to Wilson s $Q_3 T_3$ type with an initial elevation of the S T junction in Lead 3 eventually developing a downward T_3 (Figs 63 and 65) and frequently also showing a large Q_3 *or* QS_3 are obtained from cases which are found at autopsy to have an infarction in the posterior or diaphragmatic portion of the left ventricle with or without involvement of the apex This represents the usual area supplied by the posterior descending branch of the right coronary artery Likewise records of the $Q_1 T_1$ type with modifications of T in Lead 1 (Figs 64 67 and 68) and often with a small or large Q in this lead will show infarction in the anterior portion of the left ventricle possibly also involving the apex or sometimes in the apex alone This is the usual area supplied by the anterior descending branch of the left coronary

In the *precordial leads* also the S-T junction and T wave are affected by myocardial infarction and the changes are similar to those seen in the leads from the extremities The direction of the abnormal deflections of S T and T in the precordial leads is usually the same as the direction found in Lead 1 With anterior

during systole of the current of injury at the boundaries of the infarcted area The later T wave changes are probably the result of a prolongation of the duration of the contraction in the injured myocardial area *The QRS changes later to be described* and which are more permanent are probably due to the absence of the electric forces that were normally produced by the infarcted muscle

Changes in the QRS group Wilson and his associates have *pointed out that records showing the elevated S T segment* and later the coronary T wave in Lead 1 frequently also show a definite conspicuous and rather broad Q wave in this lead and a deep S wave in Leads 2 and 3 The QRS group of these records has its smallest deflections in Lead 1 (Figs 64 and 67) and often *as in Figure 64 there is a low voltage of QRS manifested by low amplitude in all leads* They also re emphasized the authors previous observation that records showing the elevated S T seg ment and later the coronary T wave in Lead 3 frequently showed a large Q wave in this lead They pointed out that some *times there is no R wave in this lead, as in Figures 63* B and C and 65 C so that the QRS group consists solely of a wide down ward deflection QS which they called Q because they believed its significance was the same as that of Q In records with large Q_3 or with QS_3 there is frequently also a Q wave in Lead 2 Thus *lead usually shows the smallest QRS deflections of any* of the limb leads (Fig 65 C)

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which may have its upward portion quite definitely convex and so resembles the coronary T wave This is seen in Leads 4 F and CF 5 of Figure 68 A while more typical coronary T waves are seen in Leads EF 3 4 F and CF 5 of Figure 68 B which represents a later stage of healing

The inverted T wave often increases in amplitude during the process of healing of the infarct and may reach a very large size measuring over 15 mm as seen in Lead 4 F of Figure 68 B With further progress of healing the amplitude gradually diminishes and the wave may become isoelectric and perhaps eventually upright

The QRS group in the precordial leads undergoes characteristic changes as a result of infarction of the anterior surface of the heart The most important feature is the disappearance of the R wave which is most likely to occur in leads from the sternal and parasternal positions less at the apex The QRS group usually consists of a large QS deflection but this deflection may sometimes be of small size in Lead 4F as in Figure 67 The disappearance of the R wave is believed to be due to the necrosis of the underlying muscle Leads from the apex may show a large Q and small R and possibly also an S wave as in Figure 68 Such complexes have been observed in experimental work at the margin of the infarcted area or when necrotic muscle is covered by a layer of relatively undamaged muscle beneath the pericardium In precordial leads as in leads from the extremities the QRS group is less liable to return to normal than is the T wave so that the absence or unusually small size of R in one or more precordial leads may be the sole residual sign of a healed anterior infarct This would be especially important if found in a lead from the apex where R is ordinarily large

With infarction of the posterior or diaphragmatic portion of the ventricles precordial leads are not as likely to show changes as when the infarct is anterior but nevertheless in certain cases they may show more definite changes than do the limb leads The features which appear are usually similar to those characteristic of Lead I in these cases that is a downward displacement of the S T junction and segment the T wave itself remaining upward (Fig 69 A) Though the changes of the S T junction

infarcts the T wave changes are usually much more definite in the precordial leads than in the leads from the extremities Rarely the opposite is the case The characteristic appearance of

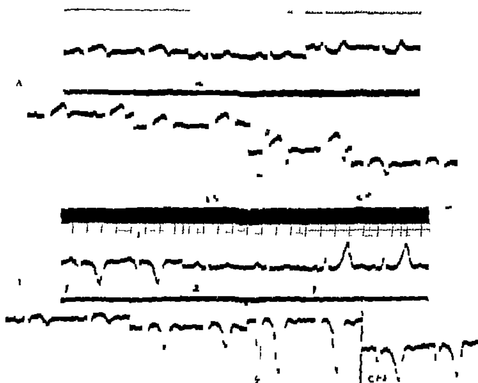


Fig 68 Two stages in the development of the change in the precordial leads record from the same patient

A This record obtained shortly after the infarct shows the Q 1 T 1 type of complex with S T elevation in Lead 1 and depression in Lead 3 In the precordial leads R is absent at the parasternal line and is small at the apex where there is a large Q There is also a large Q in Lead CF 5 T is diphasic (+ −) in all precordial leads and is of the coronary type at the apex and beyond

B Obtained ten days later shows a large Q and coronary T wave in Lead 1 In the precordial leads QRS is as in record A The T wave is of the coronary type *in Leads IF 2 IF 3 and IF and in the last of these shows large amplitude*

the precordial leads with anterior infarction shows a pronounced elevation of the S T junction and S T segment usually more marked in records from near the apex than from near the sternal border (Fig 68 A) The more numerous the precordial points showing these T wave changes the more widespread we would expect *to find the infarcted area With the progress* of healing the S T segment becomes less elevated and the last portion of T dips below the zero level to form a diphasic T wave (+−)

which may have its upward portion quite definitely convex and so resembles the coronary T wave This is seen in Leads 4 F and CF 5 of Figure 68 A while more typical coronary T waves are seen in Leads FF 3 4 F and CF 5 of Figure 68 B which represents a later stage of healing

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With infarction of the *posterior* or diaphragmatic portion of the ventricles precordial leads are *not as likely* to show changes as when the infarct is anterior but nevertheless in certain cases they may show *more definite* changes than do the limb leads The features which appear are usually similar to those characteristic of Lead 1 in these cases that is a downward displacement of the S T junction and segment the T wave itself remaining upward (Fig 69 A) Though the changes of the S T junction

infarcts the T wave changes are usually much more definite in the precordial leads than in the leads from the extremities. Rarely the opposite is the case. The characteristic appearance of

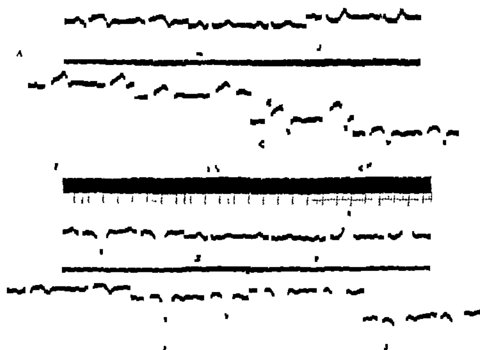


Fig. 68. Two stages in the development of the change in the precordial leads records from the same patient.

A. This record, obtained shortly after the infarct, shows the Q 1 T 1 type of complex with S T elevation in Lead 1 and depression in Lead 3. In the precordial leads R is absent at the parasternal line and is small at the apex where there is a large Q. There is also a large Q in Lead CF 5. T is diphasic (+ –) in all precordial leads and is of the coronary type at the apex and beyond.

B. Obtained ten days later, shows a large Q and coronary T wave in Lead 1. In the precordial leads QRS is as in record A. The T wave is of the coronary type in Lead CF 2, CF 3 and CF 4, and in the last of these shows large amplitude.

the precordial leads with anterior infarction shows a pronounced elevation of the S T junction and S T segment, usually more marked in records from near the apex than from near the sternal border (Fig. 68 A). The more numerous the precordial points showing these T wave changes the more widespread we would expect to find the infarcted area. With the progress of healing the S T segment becomes less elevated and the last portion of T dips below the zero level to form a diphasic T wave (+–)

that of the precordial leads Whether esophageal leads will prove to be more sensitive for the determination of infarcts on the diaphragmatic surface of the heart than are the precordial leads must develop with further experience The proximity of the esophageal electrode to this portion of the heart would make such a result likely

In certain cases the infarct may involve *both the anterior and the diaphragmatic surfaces* of the heart and thus leads to a combination of the electrocardiographic effects of anterior and posterior infarcts In the acute phase the S T segment is elevated in all three leads the elevation being most marked in Lead 2 A large Q_3 may be present In precordial leads from the neighborhood of the apex the R wave is small or absent and there is elevation of the S T segment With healing these cases may develop a normal appearance of the T wave in the limb leads while showing the absent R and diphasic T of anterior infarction in the precordial leads Other cases may develop the coronary T changes in Lead 1 or in Lead 3 during the process of healing It will be noted that the diagnostic differences between these curves and those which have been associated with pericarditis (see page 281) are that the latter do not show a large Q_3 usually have the S T changes more marked in Lead 1 and have the R wave present in the precordial leads It would seem though that in certain cases the effects of these two lesions upon the electrocardiogram might be so similar as to make differentiation difficult

Infarction in the *lateral wall of the left ventricle* also may be recognized by its electrocardiographic features A record from such a case is seen in Figure 70 Wood Wolferth and Bellet have described the characteristic features of these curves as being (1) A depression of the S T segment in Leads 1 and 2 without the elevation of the S T segment in Lead 3 ...

... marked in ... from the cardiac apex or to the left of this point The electrocardiographic features of the acute lesion in this situation tend to subside very rapidly and the tracing obtained during the process of healing may not show anything resembling the

occasionally may be most marked in the record obtained near the sternum they are usually most marked in the region of the apex With healing the S T segment returns to normal and the



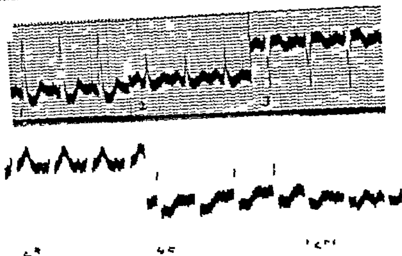
Fig 69 A A record of the Q 3 T 3 type shortly after infarction affecting the diaphragmatic surface of the heart There is slight depression of S T 1 and slight elevation of S T in Leads 2 and 3 There is a large Q 3 The precordial leads show a depression of the S T junction and a diphasic (− +) type of T wave

B A record during the healing stage probably the result of an infarct of the left lateral wall toward the apex There is an inverted T 1 and T 2 T 1 being slightly diphasic (+ −) and of the coronary type The precordial leads show a very large upward T wave in Lead II 2 a diphasic (+ −) T wave in Lead EF 3 and a downward T wave at the apex and beyond The R wave is present with all positions of the electrode

T wave itself tends to become large sometimes measuring as much as 15 mm or more as in Lead IT 2 of Figure 69 B With further progress of healing it returns to a more normal size The QRS group after infarction of the diaphragmatic portion of the heart usually retains the R wave because the degenerated muscle areas are not directly beneath the electrode

The *esophageal leads* have been shown by Hamilton and Nyboer to be affected by cardiac infarction in a manner similar to

four or forty eight hours later would in all probability show a further development of these changes or their disappearance Such variations in themselves are diagnostic of an acute myo-



I v A record and ... record of ... In Leads I and ? the S T junction ... leads the S T junct on is depress ... beyond R is present with all pos ons of the precordial electrode

cardial condition and if combined with a proper clinical history may be recognized as a result of myocardial infarction

There are a few cases of coronary thrombosis in which the electrocardiogram may not show any changes at first the appearance of these changes being delayed for three or four days in rare cases for as much as a week During this interval however there will usually be found some suggestive although not typical peculiarity of the curve Repeated records will eventually show typical changes Precordial leads are more likely to indicate the presence of anterior infarction than are the standard leads but with posterior infarction the reverse is usually the case

Bundle branch block Coronary thrombosis may also lead to the appearance of curves characteristic of bundle branch block These are somewhat more common in cases with infarction of the posterior or diaphragmatic surface because the artery supplying this region of the myocardium usually a terminal branch

coronary T wave but may more closely resemble the curves commonly found with hypertension having a depressed S T junction and inverted T in Lead 1 as in Figure 17 D These cases are particularly liable to develop auricular fibrillation at some time during their course As the infarcted area heals the deviation of the S T segment disappears and tends to leave a T wave in Leads 1 and 2 which if inverted is not in any way characteristic or which may even be upright so that no trace of the infarct may remain

Atypical changes with coronary occlusion Although in many cases of coronary thrombosis these typical changes in the QRS group and T wave appear and are seen to progress with healing as has been described yet it is not infrequent to find changes in the T wave of the same general character that is a deviation of the S T junction a deformity of the curve of the S T segment or a change in the amplitude of the T wave which are not marked enough to be typical but nevertheless follow a gradual progressive course of changes perhaps ending with a typical coronary T wave Less frequently changes in the QRS group also appear in these cases so that R_1 or R_3 may become smaller and Q_1 or Q_3 larger and yet do not develop into the typical appearance In a patient who has had an attack suggesting coronary thrombosis the appearance of any of these imperfectly typical changes in QRS or T may be taken to indicate that infarction has occurred provided that progressive changes are seen in the T wave The reason that the changes are not typical in such cases may be that the infarct is of relatively small size or is in a situation which does not exactly coincide with that usually occupied by an anterior infarct or a posterior or a left lateral wall infarct

Sometimes the changes in the precordial leads are much more definite than those found in the limb leads and it is for this reason especially that leads should be obtained from more than one precordial point Sometimes only one or two of the precordial leads show an abnormality which is recognizable the curve in the other leads being slightly unusual in form but not definitely recognized as abnormal Should a record be obtained which showed only changes which could be characterized as unusual but not definitely abnormal another record twenty

With bundle branch block one may always be in doubt as to whether or not it might have been due to a chronic process that existed before the attack. At times it is possible to observe changes in the S-T segment of a bundle branch block curve similar to those that appear in the normal curve after recent infarction, that is, an elevation of the S-T segment in Lead 1 and a depression in Lead 3 or vice versa. This effect of the infarction is superimposed on the features of the bundle branch block complex modifying the T wave in the direction characteristic of the infarcted area. In Figure 71 A such a modification of the complex of right bundle branch block can be plainly discerned. There is an elevation of S-T in Lead 1 and a depression in Lead 3, and the precordial leads show the characteristic S-T elevation with all four positions of the electrode. There is a large Q in Leads EF 2 and EF 3, and the R wave which is usually found in Leads 4 F and EF 3 of curves with right bundle branch block is not present in this record. With a posterior infarct and left bundle branch block one may have difficulty in being certain that both are present, since the S-T segment deviation of the two conditions is in the same direction. Nevertheless, as in Figure 71 B, one occasionally observes an appearance so characteristic as to be readily recognized. Bundle branch block was not present during the taking of Lead 2 of this record and the characteristic Q and S-T segment changes can be plainly seen. Lead 1 shows a depressed S-T junction while in Lead 3 it is elevated. Complete heart block is also present. Figure 71 C is a curve of right bundle branch block modified by infarction, probably involving both anterior and diaphragmatic surfaces, as suggested by the elevated S-T segment in all three leads. Figure 71 D is a later record of the same patient showing inverted T waves in Leads 1 and 2, indicating that the anterior or left lateral region shows the residual damage.

Barnes shows three instances in Figure 26 C, F, and I of his book. The first two of these suggest anterior infarction, one with left, the other with right bundle branch block, while the last suggests posterior infarction with left bundle branch block. These three cases were proven by autopsy to have the infarct in the region indicated by the electrocardiogram.

of the right coronary, sends off a branch to the auriculoventricular bundle and its ramifications Auriculoventricular heart block also is relatively common in association with infarction of the

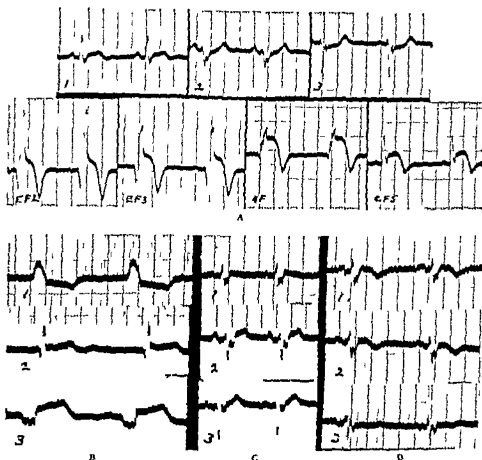


Fig 71 A Right bundle branch block type 1 with infarction of the anterior surface of the heart Note the elevation of S T in Lead 1 and depression in Lead 3 also the elevation of the S T junction in all precordial leads the large Q in Leads EF 2 and EF 3 and the delayed R wave in these leads and at the apex

B Left bundle branch block with infarction of the diaphragmatic surface of the heart Bundle branch block was not constantly present in this record and is only shown in Leads 1 and 3 Note depression of S T junction in Lead 1 and elevation in Leads 2 and 3 Note also Q 2 and elevation of S T 2 associated with a more normal intraventricular conduction

C Right bundle branch block type 1 with infarction of both anterior and diaphragmatic areas of the ventricles Note elevation of the S T junction in all three leads and absence of the usual upright T 1 found in such record of bundle branch block This is because T 1 is becoming inverted

D A record of the same patient eleven days later Note downward T wave in Leads 1 and 2

diaphragmatic surface but usually subsides with healing of the lesion

T wave and the associated changes in the QRS group such as large Q_3 who have never been subjected to an attack which could have been due to coronary thrombosis In these patients we believed the arterial narrowing to have been of slower progress taking place so gradually that degeneration and healing have gone hand in hand The electrocardiogram obtained represents the present state of such processes

If these patients are followed it will be found that in some the form of the electrocardiogram even though abnormal may remain unchanged for many months or even years In others however changes in the form of the S-T segment and in the height and the direction of the T wave may take place over relatively short periods such as several days or perhaps weeks In such cases the changing electrocardiogram indicates a changing state of the myocardium probably with healing predominant if the form is progressively more normal probably with degeneration predominant if it becomes progressively more abnormal Caution should be used however in attaching too specific a significance to the character of these changes for *we are not* certain that a change of the T wave to a more normal form always indicates that the heart muscle which was giving rise to the abnormality has become regenerated It is possible that muscle having become entirely dead may no longer affect the electrical processes of the cardiac contraction and that the T wave may then become more normal If a large myocardial area gives place to fibrous tissue it is likely *that there* will be a change in the QRS group and possibly also in the T wave resulting from the absence of the electrical effect of the muscle which has been destroyed Such a change would naturally be a permanent *one* and would not be subject to variations from time to time

Whenever we meet with a record showing an unusual degree of deviation of the S T junction with accompanying changes in the S T segment we may *conclude that* there is present an area of myocardium containing *necrotic* fibers such as are found in an infarct We cannot on the other hand be sure of the absence of such necrotic fibers *because of* the absence of the S T changes We do not know how *much* degeneration is necessary to give rise to these changes in the curve and it is likely that degenera

Relation of infarct to muscle bundles Robb and Robb have suggested a method of localization of cardiac infarcts which was correlated with experimental records obtained from dogs after occlusion of the arteries supplying particular muscle bundles of the heart Many of their illustrations from clinical cases do not show the diagnostic S T segment deviations to a degree which could be clinically diagnostic the deviations to which they call attention are often only a fraction of a millimeter in amplitude It does not seem that these small variations which are well within the limits of the normal range can properly be used as a basis for an electrocardiographic diagnosis Further the anatomical description of the myocardial areas involved frequently suggests that parts of more than one muscle bundle were involved There is rarely a discreet involvement of one muscle bundle in their cases The point of view that the individual cardiac muscle bundles *may have a specific effect upon* the electrocardiogram is one that should not be lost sight of however as it may be capable of further development These authors have indicated that a lesion of the superficial sinospiral muscle will produce an S T deviation which is upward in all three leads (+++) *The superficial bulbospiral will produce* an S T deviation which is downward in Lead 1 and upward in Leads 2 and 3 (−++) A deep sinospiral lesion will produce deviations which are upward in Lead 1 downward in Leads 2 and 3 (+−−) The deep bulbospiral lesions give rise to a low voltage *of QRS with very high origin of the S T segment in all three leads* (+++) In this connection attention should be called to the recent pathological studies by Lowe which indicate that in certain human hearts at least the abnormal myocardial areas may be limited to the distribution of one of the muscle bundles

PROGRESSIVE CORONARY OCCLUSION

The characteristic changes of the S T segment and the T wave which result from myocardial infarction have been described and the usual course of the changes observed in serial electrocardiograms of such patients has been indicated Many patients however are found to show the characteristic coronary

cardiogram of a normal heart may show slight changes The S T junction may be displaced as much as 1 mm the T waves tend to decrease in amplitude Partial or complete reversal of the direction of T_1 without any S T displacement in this lead is a rare finding but partial or complete reversal of the direction of T or T_3 or both sometimes associated with slight S T displacement was observed in about one third of the normal subjects

When patients with coronary arteriosclerosis breathe this oxygen poor mixture for a certain length of time more or less striking changes may be observed in the electrocardiogram in many cases and it is believed that these changes occur because branches of the coronary arteries are so narrowed that they are unable to bring an adequate supply of blood to the myocardial areas which they supply In other words it is believed that coronary insufficiency is the cause of these electrocardiographic changes The changes observed are as follows (1) a change in the level of the S-T junction of more than 1 mm in any lead (2) partial or complete reversal in the direction of T_1 when associated with even as little as 0 5 mm displacement of the S T junction in this lead (3) complete reversal in the direction of T in Lead 4F (4) partial reversal in the direction of T in Lead 4F associated with even as little as 0 5 mm displacement of the S T junction in this lead Similar changes in the electrocardiogram after induced generalized anoxemia were observed in five cases of severe anemia although in none of the cases studied was the S T junction displaced more than 1 mm

There is an inconstant relation between the appearance of these abnormalities of the curve and the patient s complaint of anginal pain The induced anoxemia may change the curve without causing pain in some patients while in others pain may appear without the changes being present As a test of coronary function the procedure bears promise but is still in the stage of experimentation Definite conclusions as to the absence of coronary disease cannot at present be drawn However if anemia is not present the appearance of the changes described may be taken to indicate a deficient coronary flow

tive changes more readily affect the curve when in one part of the muscle than when in another

EXERCISE

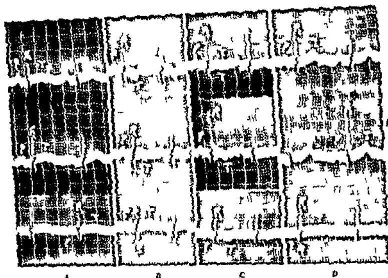
The effects of exercise upon the electrocardiogram of normal individuals have been noted by several observers and seem to be limited to relatively minor changes The P waves are increased in amplitude especially in Leads 2 and 3, and the P R level is depressed to a maximum of 1 mm as is also the level of the S T junction The T wave in some patients may be increased slightly in voltage, but it is usually slightly diminished When the exercise is exhausting the electrical axis of QRS tends to deviate toward the right

When the coronary arteries are diseased so as to be narrowed and a state of coronary insufficiency is present exercise may produce striking and characteristic changes in the S T segment and the T waves of the electrocardiogram in both the limb leads and the leads from the precordium Such changes as marked depression or elevation of the S T junction an increase in the depth of an inverted T wave or a definite inversion of a flat T wave may be produced These electrocardiographic changes are somewhat independent of whether or not the exercise produces pain They are similar to those which have been observed during spontaneous or induced attacks of angina pectoris and are not always evident immediately after exercise sometimes coming on during the following three or five minutes These changes resulting from exercise in patients with coronary insufficiency may sometimes last for as much as a half hour gradually disappearing during this time They do not usually last for more than five or ten minutes

ANOXEMIA

Several authors have studied the effects of anoxemia upon the electrocardiogram but the earliest experiments were accompanied by rebreathing of the expired carbon dioxide and were of necessity of relatively short duration Recently Levy has studied the effects of inhaling a mixture of 10 per cent oxygen and 90 per cent nitrogen without rebreathing and has found that the electro

~2) This feature may be followed later by the development of a coronary T wave. A few patients will show an unusually large Q wave in Lead 3



A B C D

Fig 2 The changes found in acute rheumatic myocarditis.

A This record shows elevation of the S T junction In Lead ~ it is slightly more than 1 mm above the baseline There is also a peculiar appearance of the S T segment this being shorter and less concave than usual On recovery this patient had a normal appearing S T interval and upward T wave in all three leads

B There is a slight degree of left axis deviation but the definitely abnormal feature is the inverted T wave in Leads 2 and 3 In Lead 2 the S T segment curves slightly upward before turning downward to the peak of the T wave suggesting but not duplicating the coronary T

C

cc n

D

the [illegible] at recovery had taken place three [illegible] after record C was obtained and is a normal record

The T wave of the precordial leads likewise is affected by the rheumatic myocardial process either coincident with changes in the T wave of the leads from the extremities or independently so that in some patients the precordial leads alone will show abnormal features The T wave may be unusually large in some cases in others it may be unusually small it may be diphasic (+ −) or (− +) or notched it may show the coronary T form or may simply be inverted These T wave abnormalities vary from time to time during the course of the disease and often persist

Dissecting Aneurysm

Since the clinical syndromes of dissecting aneurysm and coronary thrombosis may resemble one another, it is important to *note that the electrocardiogram in the former condition may* sometimes be found abnormal The abnormality is due to the effect upon the myocardium of a coincident arteriosclerosis of the coronary arteries so that any variety of abnormal ventricular complex may be found which might be expected in this disease If the coronary arteries are seriously narrowed the fall in pressure which accompanies dissecting aneurysm will sometimes result in such a degree of myocardial anoxemia that changes in the S T segment may appear resembling those associated with infarction but of relatively slight degree *Since cardiac infarction* itself usually results in typical changes within the first few hours *the finding of a normal electrocardiogram in a suspected case or* even of an abnormal one which is not typical of infarction should be considered in an appropriate case as evidence in favor of a dissecting aneurysm rather than infarction being present

Acute Rheumatic Fever

If frequent records are taken during the acute stage of this disease more than 60 per cent of cases will be found to show inversion or other abnormalities of the T wave such as have been described as resulting from myocardial disease A larger number will show variations in the duration of the auriculoventricular conduction time indicating a *myocardial process in the* A V tissue which may go on to the production of dropped beats and occasionally to complete heart block Many will show both of these types of electrocardiographic change

Many of the patients who do not show inversion of T will be found to show minor variations from the normal form of the T wave These especially affect the voltage of T which may become smaller and the form of the curve of the S T segment so that this may run straight to the peak of T or may be convex upward giving rise to a broad topped rounded T wave A few patients especially those with evidence of pericarditis may show elevation of the S T segment in Leads 1 and 2 similar to that seen in Figure

tricular complexes either notching of QRS an abnormal inversion of T or both Occasionally bundle branch block will be present As a rule the excursions of the waves are of fairly good size perhaps because of the hypertrophy which is present at this stage of the disease A myocardial abnormality plus the valvular disease constitutes a considerable handicap so that these patients are not so likely to recover their former ability under treatment as are those with only the valvular handicap

Certain patients with chronic inactive valvular disease will show a prolongation of the auriculoventricular conduction time which would not have been suspected without a polygraphic or electrocardiographic record This abnormality does not add greatly to the gravity of the prognosis for the disease does not usually progress to the higher grades of block but if we know of its presence we shall be prepared for the appearance of dropped beats upon thorough digitalization

Pericarditis

Electrocardiographic changes will be found during the course of pericarditis in a considerable proportion of cases if records are taken frequently during the disease Certain changes are associated with the acute inflammatory stage of the disease and these are of relatively short duration frequently quite transient Bellet and MacMillan report that they have never observed these changes to persist in the limb leads for more than twelve days although the precordial leads may continue to show the abnormality for several days after it has disappeared from the limb leads The changes usually consist of an elevation of the S T junction in Leads 1 and 2 occasionally Lead 3 may be similarly affected (Figs 72 A and 73 A) In certain records elevation of S T may only appear in Lead 1 The S T segment may be concave upward in passing to the peak of T or it may be a straight line or in some cases it may be convex upward so that T as a whole has a dome shaped contour as after myocardial infarction

Precordial leads show similar changes in the S T segment which is elevated above the isoelectric level (Fig 73 A) The R wave is present in almost all cases Bellet and MacMillan observed that the most markedly characteristic changes in the pre

after the T wave in the limb leads has already returned to normal

As the acute phase subsides these electrocardiographic abnormalities usually disappear giving place to a normal record Occasionally however they may persist for a considerable time after other acute manifestations of the disease have subsided and it is probable that this may indicate that the acute process is still active in the heart It is rare for these changes to be permanent but when this occurs it is likely that a fibrotic change has taken place in the myocardium One must remember in connection with such cases that rheumatic myocarditis may maintain a low grade of activity over long periods of time and a decision whether an abnormal electrocardiogram represents an active or an inactive process is sometimes impossible without an opportunity to obtain serial records Should variations appear in serial records, it would be an indication of a changing state of the myocardium, which usually would be due to a continuation of an active process occasionally to the fibrosis which might follow after the subsidence of an acute process Here as is usually the case the decision as to the interpretation of the electrocardiographic changes must be made on clinical grounds

The appearance of electrocardiographic abnormalities in acute rheumatic fever is independent of the appearance of valvular disease of the heart Acute valvulitis occasionally will be present without any change in the electrocardiogram but it is not uncommon for the electrocardiogram to be affected without the valves It is because the acute rheumatic condition commonly involves so many different parts of the heart that the signs of valvular and myocardial change usually occur together

Rheumatic Valvular Disease

Patients with chronic rheumatic valvular disease without signs of activity of the rheumatic process do not often show significant abnormalities of the waves of the electrocardiogram If however these patients have symptoms referable to the heart, either of palpitation or of shortness of breath or pain abnormalities of the curve are much more liable to be present At this stage about one in ten will show some significant abnormality of the ven

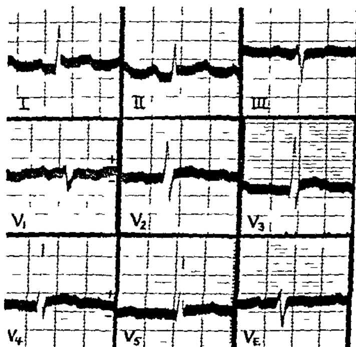
tricular complexes either notching of QRS an abnormal inversion of T or both Occasionally bundle branch block will be present As a rule the excursions of the waves are of fairly good size perhaps because of the hypertrophy which is present at this stage of the disease A myocardial abnormality plus the valvular disease constitutes a considerable handicap so that these patients are not so likely to recover their former ability under treatment as are those with only the valvular handicap

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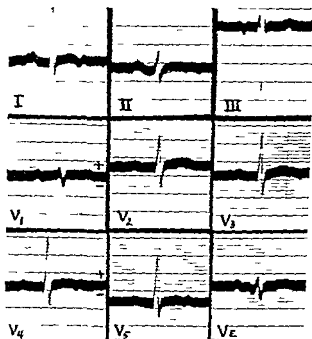


Fig 73 From a patient with acute rheumatic pericarditis Leads 1 2 and 3 are indicated by the Roman numerals the letters V_1 V_2 V_3 V_4 V_5 and V_E indicate leads made with the precordial electrode placed upon the first five positions recom mended by the American Heart Association the point *E* is over the ensiform

cordial leads were obtained when the electrode was placed over an area where pericardial friction was heard

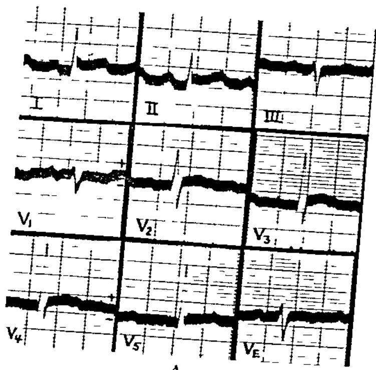
This acute phase marked by elevation of the S T junction may be followed by a return of the electrocardiogram to normal Usually however the later part of the T wave comes to dip below the zero level so that the wave is now diphasic (+−) and the peak of the upward S T segment being rounded and upwardly convex together with the downward peak which follows it produces the form of the coronary T wave as in Leads 1 and 2 of Figure 73 B This usually appears most prominently in the leads which have shown the most prominent deviation of the S T segment It may involve Lead 1 or Leads 1 and 2 and occasionally all three leads It may also appear in one or more of the precordial leads as in the figure where it may persist for some time after the T waves of the limb leads have become normal A peculiar appearance is occasionally noted in which the S T segment taking origin at or slightly above the isoelectric line first curves upward and then turns almost at an angle to pass sharply downward to the peak of an inverted T Such T waves are also observed following myocardial infarction (Lead 2 of Fig 69 A and Lead 1 1 3 of Figure 69 B) As recovery progresses the T wave will usually return to normal though there may be a residual inversion of T which shows no special characteristics Cases of pericarditis which fail to show these characteristic S T segment and T wave changes are usually found to be due to a chronic inflammatory reaction such as tuberculosis

When there is marked pericardial effusion low voltage of the T waves and possibly also of the QRS group may be found Low voltage T waves have often been observed in cases of constrictive pericarditis The precordial leads in such cases do not

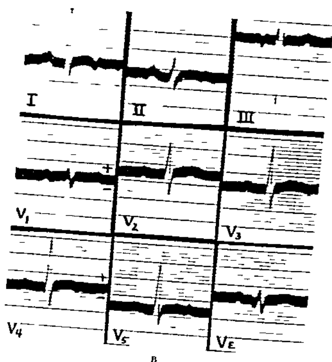
[illegible] place I indicates that the central terminal recommended by Wilson was used as a [illegible] Time lines are [illegible] second and the precordial leads [illegible] standardized so that 0 0 [illegible] cm equals 1 millivolt (These records were kindly [illegible] by Dr Charles E [illegible])

A During the acute stage showing elevation of the S T junction in Leads 1 and 2 [illegible] inclusive In these leads the T wave is notched

B Fifteen days later showing the coronary T wave in Leads 1 and 2 The [illegible] of the S T junction in the precordial leads is less marked and in [illegible] there is a slight downward deflection before the end of T so that the [illegible] form is simulated



A



B

Fig 73 From a patient with acute rheumatic pericarditis Leads 1 2 and 3 are indicated by the Roman numerals the letters V_1 V_2 V_3 V_4 V_5 and V_E indicate leads made with the precordial electrode placed upon the first five positions recommended by the American Heart Association the point E is over the ensiform

plete heart block and is often complicated by bundle branch block has a very high mortality and usually develops very suddenly with severe clinical symptoms It should be emphasized that the appearance of arrhythmia during the acute stage of diphtheria should suggest the possibility of a conduction defect being present but even in the presence of a conduction defect a regular idioventricular rhythm of 80 to 90 per minute may occasionally be present giving no suggestion of the underlying disease

Lobar Pneumonia

In about 20 per cent of cases of lobar pneumonia one finds inversion of the T wave either in Leads 2 and 3 or in all three leads The inversion is sometimes accompanied by an upward convexity of the S T segment so that there is a resemblance to the coronary T wave These changes are much more frequently found in cases that have a fatal outcome In cases that recover the T wave may return to normal within two or three days or it may not become normal for four or five weeks Occasionally the T wave will be of low voltage and this is followed *in some* cases by inversion of T Changes in the S T segment are also observed During the acute stage these may resemble the appearance characteristic of pericarditis and it is likely that they indicate the presence of this complication If the T wave is *inverted it* may resemble the coronary T as has been mentioned Alternation of the QRS complexes has been observed and not always in fatal cases During convalescence a slight upward deviation of the S T junction may appear At this time there may also be prolongation of the P R interval and bradycardia with an increase in the voltage of the T wave

In nine fatal cases studied at autopsy Master and Romanoff reported pericarditis in one with an electrocardiographic curve typical of the condition this case also showed numerous small foci of parenchymatous degeneration of the myocardium The other eight cases all showed changes in the S T segment which were not described in detail and the microscopic examination showed slight moderate or severe parenchymatous degeneration

In addition to the changes in the ventricular complexes pre

show low amplitude but may show notched, diphasic or inverted T waves

Diphtheria

During diphtheria the heart may be very seriously affected with resulting electrocardiographic changes Burkhardt Eggleston and Smith found that this occurs in 26 per cent of all patients suffering from this disease and it is not prevented even by the early administration of large doses of antitoxin The incidence *of peripheral nerve palsies in these patients is very high* The most frequent electrocardiographic changes consist of alterations in the T wave which occurred in 16 per cent of Eggleston s series They usually appeared during the second week of the disease though sometimes as early as the fourth day or as late as the fortieth The earliest changes are a slight depression of the S T segment and a decreased amplitude of the T wave As *the cases are followed, the T wave may become progressively* smaller until it becomes isoelectric or diphasic (—+) or (+—) and finally inverted During the diphasic stage the coronary T wave may be simulated These T wave changes occur in all three leads but are most marked in Lead 2 With recovery the T wave may return to normal which occurs in the average case in about three weeks but exceptionally may take as much as ten weeks Many of these patients with T wave changes *do not* show definite symptoms referable to the heart and this is particularly so during convalescence when there may not even be an abnormal tendency to tachycardia There is not a serious mortality rate in patients who show T wave changes in marked contrast to the group showing conduction defects

Conduction defects are not quite as frequent an occurrence as the abnormal T wave being found in about *12* per cent of cases and on the average develop somewhat earlier in the course of the disease than do the changes in the T wave A few patients who have originally shown T wave changes may later develop conduction defects Bundle branch block or other evidence of defective conduction in the bundle branches may appear and is a serious complication having a high mortality Auriculo ventricular dissociation which *usually* takes the form of com

present throughout the heart muscle and the electrocardiogram may be abnormal in one or more of the ways described in Chapter IV Simple inversion of T_1 or T may be seen in occasional cases of subacute bacterial endocarditis

Syphilis of the Heart

Syphilitic aortitis and valvular disease are frequently associated with myocardial degeneration which causes abnormalities of the electrocardiogram This is especially liable to occur when the disease involves the aortic valves The proximity of these structures to the mouths of the coronary arteries may lead to a narrowing of the orifices so that the blood flow to the myocardium is diminished

Electrocardiographic abnormalities are not specific for this condition but may be of any of the types described in Chapter IV Arrhythmia may also occur but is not common With aneurysm of the aorta the electrocardiogram is usually normal unless the aortitis has also involved the region about the mouths of the coronary arteries

Chamberlain and Fellows and also Arnett found electrocardiographic changes only in the tertiary stages of the disease The frequency of their occurrence in the 522 syphilitic patients of these two reports was about 1 per cent Bundle branch block curves were found in three patients inversion of T_1 or of T_2 in eight and prolonged auriculoventricular conduction time in two On the other hand among fifty patients admitted to the hospital with definite syphilitic cardiovascular disease Juster and Pardee found abnormal electrocardiographic features in 78 per cent There was auricular fibrillation in three and prolonged AV conduction time in two Twenty showed significant abnormalities of QRS such as abnormal duration notching low voltage or large Q_3 Thirty five showed significant abnormalities of T such as inversion of T_1 or T_2 diphasic T_1 or T low voltage or coronary T

Pulmonary Embolism

The embolism of a large branch of the pulmonary artery has been observed to be followed by certain changes in the electro

mature beats auricular fibrillation and auricular flutter were occasionally observed

Trichinosis

Electrocardiographic changes have been observed during the course of trichinosis in about one third of the cases studied The changes usually appear during the second week of the disease and may persist for a month or more The most usual abnormalities are flattening or inversion of the T wave usually occurring in Leads 2 and 3 and in one of these leads the S T segment may show the upward convexity characteristic of the coronary T wave Low voltage of the *QRS* group is an occasional finding and prolonged auriculoventricular conduction time has been reported Changes have also been observed in the T wave of precordial leads a depression of the S T segment and diminished amplitude of T being found

Other Acute Infections

Abnormal inversion of the T wave and changes in the S T segment have been observed by Master and Jaffe in cases of typhoid fever typhus influenza bronchopneumonia periarteritis nodosa Malta fever and pulmonary tuberculosis Prolonged auriculoventricular conduction time has also been observed in typhoid fever typhus and influenza As with lobar pneumonia so also with typhus fever this usually occurred at the time of or shortly after the crisis

Bacterial Endocarditis

It is not usual for the electrocardiogram to be abnormal in cases of bacterial endocarditis although abnormalities occasionally result from the blocking of a coronary artery by material from the vegetations In such a case the electrocardiographic picture of cardiac infarction will appear as after coronary thrombosis Sometimes small emboli penetrate into smaller branches and give rise to abnormalities of the T wave and occasionally a mural ulceration may involve branches of the auriculoventricular bundle so that bundle branch block may be induced In cases of acute bacterial endocarditis multiple infectious foci may be

cardiogram which in one lead may slightly resemble the changes produced by coronary arteriosclerosis When considered as a whole however the changes due to pulmonary embolism are quite different from those due to coronary arteriosclerosis and seem to constitute an important aid in the differential diagnosis of these conditions Durant and his associates having obtained records within two hours after the occurrence of the embolism found an increased duration of the QRS group due to a broadening of S in Leads 1 and 2 (Fig 74 B) At the same time there is a Q wave and a broad R in Lead 3 so that the record in their two cases somewhat resembles the complexes of right bundle branch block of Bayley s type 3 This S wave rapidly becomes narrower but the S T junction remains below the zero level in Leads 1 and 2 for several hours The junction may be so gradual as to be almost an indeterminate transition Similar effects upon the S wave and S-T junction were observed after the injection of air into the femoral vein of dogs

After from six to ten hours the appearance is like that seen in Figure 74 C Such records have been previously observed and have been considered characteristic of pulmonary embolism At this time *in* Lead 1 the S wave is well developed and perhaps broadened the S T junction is at the zero level or perhaps slightly below it and the T wave upward *In Lead 2 the* S-T junction is at zero or perhaps slightly below it and T may be small isoelectric diphasic (+−) of coronary type or perhaps inverted *In Lead 3* Q is definite or may even reach quite large size The S T junction may be slightly elevated or more frequently the S T segment is convex upward and followed by a downward T as in the *coronary* T wave In the precordial lead from the apex the R wave is present although it may be of relatively small size the S T junction is normal or slightly below zero and the T wave isoelectric or inverted These changes may not remain in characteristic form for more than a few hours or may remain for as much as two or three days the duration probably depending upon the size of the artery occluded

The possibility of confusing these records taken a few hours after pulmonary embolism with the results of infarction of the



A A record made a week before the attack which except for left axis deviation and a vibratory QRS group in Lead 3 is normal

B A record made shortly after the attack which shows a QRS duration of 0.12 second increased size of S 1 with a broadening of this wave and of S 2 a definite Q 3 is present the S T junction is depressed in Leads 1 and 2 T 3 is diphasic (+ −) with the peak downward The precordial leads [illegible] and [illegible] show depression of the S T junction and inversion of T in Leads V_1 and [illegible]

C Two days later The limb leads show a return of the duration of QRS and of S 1 and S 2 to normal A large Q 3 has appeared the S T junction is at normal level [illegible] is of the coronary type and T 3 is deeply inverted The precordial leads show QRS practically returned to normal [illegible] of T in [illegible] and a normal S T junction [illegible] T is smaller than normal [illegible]

D [illegible] days after the attack QRS has practically [illegible] T 3 [illegible]

Contusion of the Heart

Contusion of the heart may occur either with or without fracture of the ribs or sternum the heart being crushed usually against the vertebral column A myocardial injury results which may give rise to abnormalities of the ventricular complex as well as to cardiac arrhythmias The most frequent changes are found in the T wave which may become inverted or be of low voltage or of the coronary type These changes are sometimes more definite in precordial leads than in the leads from the extremities They tend in time to recovery but their duration will probably be found to depend upon the extent and severity of the myocardial injury

Hypertension

Records obtained from patients with hypertension show left axis deviation of QRS in about 60 per cent of cases About 20 per cent show an increased voltage of QRS and many show an increased duration of QRS so that it may measure 0 11 or 0 12 second About 30 per cent show an abnormality of the ventricular waves the most usual being an inversion of the T wave in Lead 1 and possibly also in Lead 2 This inversion is often accompanied by a deviation of the S T junction below the zero level in Lead 1 and an elevation above it in Lead 3 (Figs 48 B and 77 E) Myocardial degeneration or interstitial fibrosis is usually present in these cases and is due to the effects of coronary arteriosclerosis Occasionally however these electrocardiographic changes may be present without there being myocardial disease Ryker and Hepburn found four out of twenty hearts with records of this sort to be free of myocardial disease and to have fairly normal coronary arteries when studied post mortem These features of the S T segment and T wave may be due to left ventricular hypertrophy as has been pointed out in Chapter III or possibly to left ventricular strain as maintained by Barnes and Whitten The precordial leads may also show abnormality of the T wave it being found either diphasic or inverted and sometimes having the coronary form (Fig 77 E) These changes are more usual in leads from the region of the apex or beyond Their relation to

diaphragmatic surface of the heart which also produces a large Q_3 and inverted T_3 of coronary type will be avoided if attention is paid to the fact that with diaphragmatic infarction it is rare to find a large S_1 and it is usual to find S T elevated in records obtained soon after the attack and unusual to find the coronary T wave early With diaphragmatic infarction the precordial lead rarely if ever, shows a downward T wave

TRAUMA

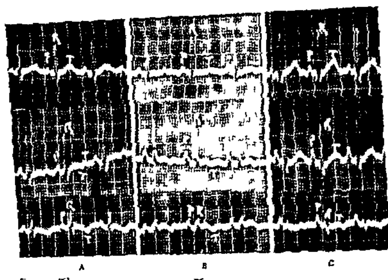
The heart may be affected by stab wounds and secondary hemopericardium may occur Stab wounds usually but not always produce an effect upon the electrocardiogram Hemorrhage into the pericardium if of considerable amount will produce such evident effect upon the blood pressure and venous pressure that the electrocardiogram should not be needed Actually records taken under these circumstances have not been published although Porter and Bigger reported that when cardiac tamponade developed in a postoperative case and was relieved by opening the pericardial wound there was no change in the previously abnormal electrocardiogram It would be expected that as with pericardial effusion the voltage of all waves would be reduced by the presence of blood in the pericardium and increased after its withdrawal

The operative repair of stab wounds has been found to result in a series of electrocardiographic changes similar to those found with pericarditis an S T elevation is present in all three leads, and the coronary T wave that appears later is best marked in Lead 1 or Leads 1 and 2 A return to a normal form of the electrocardiogram has been observed as soon as three months after the operation but in other cases abnormality of the T wave has persisted for five or six months or more In a case of Porter and Bigger a terminal branch of the anterior descending coronary was tied off but the reciprocal relationship of the S T changes characteristic of anterior myocardial infarction was not observed S T showed a typical elevation in Leads 1 and 2 in the record immediately after operation* and in all three leads three days later This is like the picture associated with pericarditis

* *Lead 3 of this record has the S T junction below the isoelectric level but the appearance is typical of the overshooting due to high skin resistance (Fig 97) rather than the depressed S T junction due to cardiac infarction (Figs 67 and 68 A)*

level like that following coronary thrombosis has not been observed with hyperthyroidism

When the toxicity passes off the T waves of these patients



phasic (+ −) T in Lead 2 s low voltage and is diphasic

c There is a large voltage of the P wave T 1 shows a peculiar straightening out of the normal upward concavity of the S T segment while T 2 shows a broadening of the peak (dome shaped)

will usually return *to normal* In long standing cases when chronic fibrotic *changes have* made their appearance we may see notching or slurring of the *QRS* groups with a typical persistent inversion of the T *wave in Lead 2* or in Leads 2 and 3 These changes being fibrotic in origin are permanent They are often in these cases *due to a complicating arteriosclerosis* of the coronary arteries and not *to the* thyroid disease

Hypothyroidism is *often associated* with a low *voltage of QRS* and a low voltage of T This is *usually the* case when the basal metabolism reaches a figure of *−20 per cent* or less and in such cases the T wave is often *inverted in Lead 1* or in Leads 2 and 3 or in all three leads The S T *segment* may show an upward convexity suggesting the coronary T wave but in contrast *to the*

underlying pathological changes is not known but probably, as with the standard leads they are usually though not always due to pathological changes in the myocardium

Thyroid Disease

When *hyperthyroidism* is present in mild or moderate degree it is rare to encounter significant abnormalities of the ventricular waves Paroxysmal auricular fibrillation occurs occasionally or *may be a permanent feature Premature beats or paroxysmal* tachycardia also may occur in these patients When hyperthyroidism is severe these arrhythmias are found with greater frequency and auricular fibrillation is more likely to be permanent In addition to this the more definitely toxic cases are likely to show peculiarities in height and form of the individual waves of the electrocardiogram The commonest abnormality is an increased voltage of P Rose Wood and Margolies found it to be over 3 mm in about one third of their series of toxic cases and found it reduced to less than 3 mm in almost all of the same cases after thyroidectomy There is also a tendency for the voltage *of QRS to be somewhat increased and with fair frequency there is* an increased voltage of the T wave

In addition to changes in the height of T changes in the form of the S T segment and T wave are not infrequent in the more toxic cases These changes may be transient and varying perhaps present in one record and absent in the next but in other cases they are constantly present After thyroidectomy however they usually disappear showing that they are due to reversible changes in the myocardium and not to structural changes The S T segment in the leads from the extremities may become straightened (Lead 1 of Fig 75 C) or convex upward (Leads 2 and 3 of Fig 75 B) or the apex of T may become broad and rounded (dome shaped) (Lead 1 of Fig 75 A) The T wave may become diphasic (+—) in Lead 2 (Fig 75 A) more rarely in Lead 1 and may resemble the coronary T wave (Leads 2 and 3 of Fig 75 B) It may become definitely inverted in Leads 2 and 3 rarely so in Lead 1 Precordial leads may show T wave abnormalities of a similar character Deviation of the S T junction from the zero

Pellagra

Pellagra is often associated with some unusual shortness of breath and occasionally the heart sounds may be feeble and slight edema may appear It is probable that these manifestations are due to a coincident deficiency of vitamin B 1 Nevertheless the two reports of the electrocardiographic findings of pellagrins have agreed in demonstrating frequent appearance of inversion of the T wave in Lead 1 or in Leads 2 and 3 occasional appearance of a T_1 of the coronary type occasional low voltage of T and occasional low voltage of QRS Feil has found a prolongation of the constant K which represents the duration of Q T in relation to the heart rate Measurements of the duration of the mechanical systole in synchronous records of the heart sounds and subclavian pulse showed that these also indicated a prolonged duration of the contraction in relation to the heart rate

Digitalis

Digitalis frequently causes a prolongation of the auriculo ventricular conduction time This effect does not appear with small doses but with larger doses may become so marked that it eventually leads to dropped beats and occasionally to complete heart block The effect of digitalis upon the P wave is not marked but consists of a diminution of the voltage of P especially in the amplitude of Lead 3 which may become inverted The effect of digitalis on the ventricular complex is characteristic The QRS group is not affected but the S T segment the T wave and the Q T interval are affected with great constancy The effect upon the Q T interval is seen in certain cases when the T wave remains unchanged

Digitalis produces a shortening of the Q T interval which is best recognized if Q T is considered in relation to the heart rate The value of the constant K which expresses this relationship (page 68) will become smaller with increasing digitalis effect The decrease in the value of K may appear as soon as one hour after the administration of 1 to 1 5 gm of digitalis or may not appear until four hours after this dose The reduction of the

usual appearance of the coronary T the voltage of T in these cases is small The auriculoventricular conduction time frequently is found prolonged

These changes probably are due to physiological variations in the muscle metabolism and not to pathological changes because the administration of thyroid extract will lead to an increase in the voltage of the QRS group and of the T wave and will abolish the T wave inversion It will likewise shorten the auriculoventricular conduction time If the records of these patients show changes of a different character or if the changes present *do not respond to thyroid medication they are probably the* result of a complicating coronary arteriosclerosis

Neurocirculatory Asthenia

Certain patients with neurocirculatory asthenia may show inversion of the T wave in Leads 2 and 3 Inversion of T_1 however has not been observed The inversion of T may vary from time to time and the T wave may occasionally be upward in *Lead 2 and downward in Lead 3 When* followed over a long period T may become constantly upright in Lead 2 Patients who have shown inversion of T and T_3 rarely come to show an upright T_3 *In* some patients the inversion of T_2 may be associated with an erect position of the patient and when this is so records taken in the reclining *position* will usually show an upward T while records obtained when the patient is seated will show a downward T

Beriberi

Beriberi and minor grades of deficiency of vitamin B 1 have been found *associated with abnormalities of the electrocardiogram* Weiss and his associates found inversion of the T wave in Lead 1 or in Leads 2 and 3 in 68 per cent of a series of sixty seven cases and the T wave was often diphasic and occasionally showed the coronary form A low voltage of QRS was occasionally observed as was also intraventricular block The Q T duration was prolonged in about one half of the cases using the formula of Shipley and Hallaran and in 80 per cent using the formula of Cheer and Li

of the T wave although this may sometimes be accompanied by a depression of the S T segment (Fig 77 B D) The S T segment becomes further depressed and the T wave lower so that eventually the T wave may have a diphasic (—+) appearance (Lead 1 of Fig 77 B) or may consist solely of a downward deflection (Lead 2 of Fig 77 D) In other cases there is a progressive diminution of the height of T until it becomes isoelectric and then inverted without there ever being a marked change in the level of the S-T segment This is seen in the serial records of Figure 76 The T wave effects of digitalis are accentuated by the administration of atropine of acetylcholine and by carotid sinus pressure These observations suggest that it is due to a vagal influence

When the T wave is abnormally directed digitalis will diminish the voltage but does not always cause further inversion (Fig 77 F and I) It may occasionally change an inverted T to an upright one In records showing right or left axis deviation digitalis usually changes the T wave in such a way that it comes to be directed opposite to the chief deflection of QRS in Leads 1 and 3 and sometimes in Lead 2 also

These effects of digitalis on the T wave and on the S T segment may be noted as soon as one or two hours after the administration of a single large dose of the drug such as 1 minim of the tincture per pound of the patient s weight A dose of 50 minims will usually produce a slight change in the voltage of the T wave by the third or fourth hour The effect upon T reaches the maximum for any one dose about six hours after the drug is given and does not diminish appreciably until after twenty four hours It passes off very slowly and traces of a marked effect persist for at least two weeks and often as long as three weeks after the drug is discontinued For this reason final conclusions should not be drawn as to whether an abnormal T wave is due to disease until after the drug has been withheld for this interval

In the precordial leads *(Fig 77)* digitalis will usually decrease the amplitude of an upward or a downward T wave but will sometimes increase the amplitude of a downward one It also tends to depress the level of the S T segment so that the T wave

value of K usually appears simultaneously with the decrease of the voltage of the T wave The maximal effect is produced between three and four hours after each dose, sometimes persists

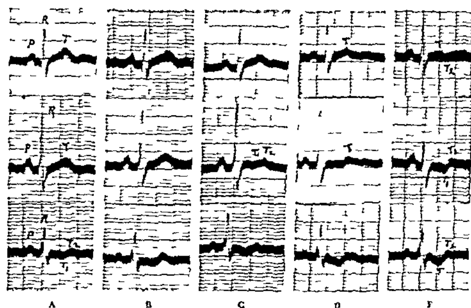


Fig. 76 Progressive increase of the digitalis effect upon the T wave Note the peculiar double topped T wave of Lead 2 record C There is a gradual decrease in the voltage of T and in records C D and E a depression of the S T junction and S T segment

for a day or so and then gradually subsides but the normal value is not reached until about a week after the last dose K returns to normal before the voltage of the T wave reaches its normal height The shortening of K seems to have a definite relation to the appearance of toxic effects When it is reduced to approximately 0 31 minor toxic symptoms usually appear when it reaches the value of 0 29 marked toxic symptoms are seen Dieuaide did not find that atropine changed the Q T duration after digitalis

Changes in the S T segment and in the T wave may be observed in almost all cases after the administration of digitalis although in occasional instances—perhaps a tenth of normal electrocardiograms—these changes may fail to appear Usually as in Figure 76 the first change is a diminution of the voltage

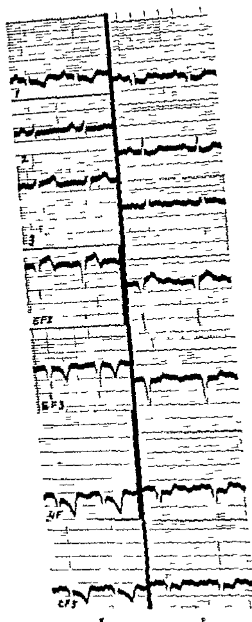


Fig. E. Record of a patient with marked anginal syndrome and moderate dyspnea on exertion. The appearance of Leads EF 2 and EF 3 is similar to that often seen during the healing stage of *infarction*. This patient had never had an acute attack. The electrocardiogram must be the result of chronic changes.

F. The same patient after digitalis showing *mild toxic* symptoms after 41 cat units in seventeen days. Note the increase in A-V conduction time which is now 0.20 second. The S-T segment is depressed in Leads 1 and 2 and this is followed by a slight upward deflection so that the wave is diphasic (− +). The voltage of T is diminished. In the precordial leads the amplitude of T is diminished in all four leads. Leads EF 3, 4 F and CF 5 show a less inverted T wave than before.

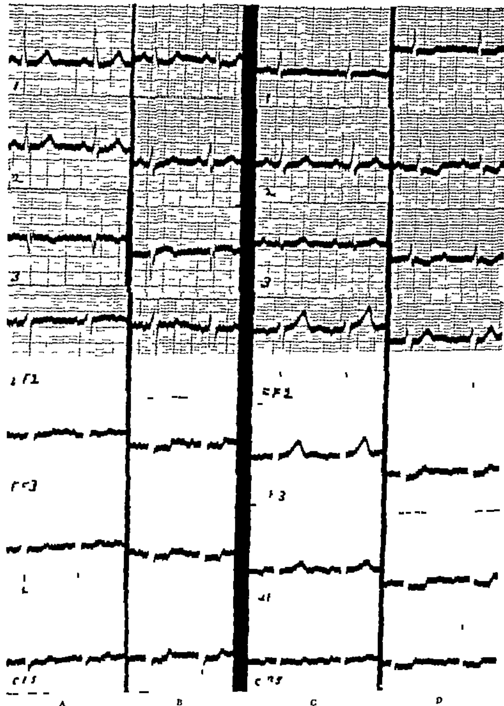


Fig 7. The records of three patients before and after the administration of digitalis

A Record of patient with obesity and hypertension before digitalis

B After digitalis 1 cat unit three times daily for two weeks Note in the limb leads the depressed S T segment in Leads 1 and 2 and the diminished voltage of T In the precordial leads note the depressed S T segment in all leads and the increased amplitude of T

C Record of patient with anginal syndrome and moderately increased dyspnea on exertion

D The same patient after digitalis 16 cat units in four days Note the prolonged A V conduction time (0 28 sec) in Lead 2 0 40 second in Lead 3 the depressed S T segment and the inversion of T in the three limb leads In the precordial leads note the depression of the S T segment in all leads and the diminished amplitude of T in Leads FF 2 and EF 3 with inversion of T at the apex and beyond

QUINIDINE

The most important effects of quinidine are exerted upon the auricles The duration of the contraction (refractory period) is prolonged and the rate of an existing circus movement is slowed by a depression of the conductivity of the muscle These effects often result in the changing of auricular fibrillation into auricular flutter or normal rhythm or in the changing of auricular flutter to normal rhythm It also diminishes the frequency of premature beats wherever they may arise probably by depressing the irritability of the muscle An effect upon the normal P wave is not always observed though there may be a change in the form of P especially an increase of its duration Sometimes the P wave takes such an abnormal form that it seems evident that the site of impulse formation must have been displaced to an abnormal focus within the auricles In many cases the auriculoventricular conduction time is increased as in Figure 78 B Large doses may lead to a prolongation of the intraventricular conduction so that the duration of QRS is increased as is also seen in the Figure The Q T duration is also prolonged The earliest and most constant effect upon the ventricular complex is a decrease in the voltage of T the amplitude of the T wave in all leads As this effect becomes more marked it leads to flattening or inversion of the T wave in one or more leads In some instances the S T segment is depressed in Leads 2 and 3

In the precordial leads also there is a diminution in the height of the T wave and a depression of the S T segment The changes observed are often as in Figures 78 B more striking than those observed in the limb leads Inversion of a previously upright T wave is more frequent in precordial leads than in the leads from the extremities

EPINEPHRINE

Experimental injection of large doses of epinephrine into animals has led to striking changes in the S T segment similar to those associated with infarction Subcutaneous injection into human beings produces only slight changes in the records from normal hearts consisting of a decrease in the voltage of the T

may become diphasic (—+) or may become entirely inverted. A T wave which was originally diphasic may become totally inverted or may increase in its positive and negative deflections.

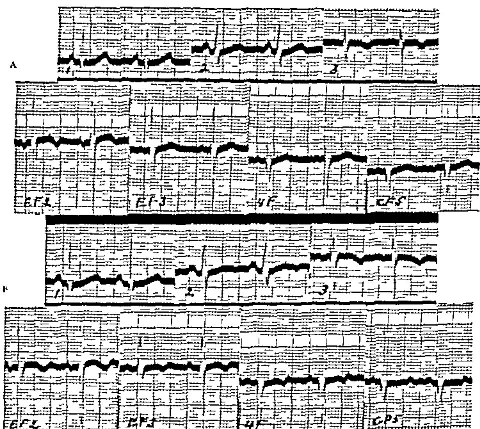


Fig. 78. Illustrating the effects of quinidine.

A. Control record of a patient who had paroxysmal attacks of auricular fibrillation.

B. Obtained after the patient had received quinidine sulphate [illegible] grams four times daily, the last dose being two hours previous to the taking of the record. The voltage of P and of T is diminished. T 2 is rounded and slightly notched. The auriculoventricular conduction time is prolonged, measuring 0.0[illegible] second more than in the control record. The QRS duration is also prolonged 0.02 second more than in the control record. In the precordial leads, note the increased inversion of T in Lead EF 2, the diphasic + — and inverted T in Leads EF 3 and 4 F, and the change in the S-T segment with diminished amplitude of T in Lead CF 5.

Very rarely there will be no change or only a very slight change in the contour of T, perhaps only a diminution in amplitude. These changes in the S-T segment and T wave are usually more marked in leads from the apex than in those from near the sternum.

Nicotine

That nicotine produces an effect upon the electrocardiogram has been amply demonstrated. The T wave is decreased in voltage and may become inverted in Lead 2 or in Lead 1. This coincides with an increase in the heart rate and blood pressure but is not dependent upon them. It occurs in 30 per cent of the subjects tested and is demonstrable not only with ordinary tobacco but also when denicotined tobacco is used and when ordinary tobacco is smoked using a filter holder. Emotionally labile individuals have been found to show these changes more frequently and to a more marked extent than those who are more stolid. It occurs less frequently and less definitely in older individuals than in those who are younger. This action of the nicotine may be due to stimulation of the sympathetic nerve fibers to the heart or to actual stimulation of the muscle fibers themselves. It is at any rate a quite transient effect, passing off in a few minutes after it has become evident. It has not been possible to demonstrate a vasoconstriction of the coronary arteries though other arteries are known to be constricted.

Insulin

In diabetic coma the electrocardiogram is often found to be abnormal. The most frequent change, found in about three fourths of the cases, is an increased duration of the Q-T interval as expressed by the factor K. The S-T segment is frequently found depressed, the T wave is often inverted and is sometimes diphasic (−+) due to a downward movement of the curve during the S-T segment. These changes are not always present in records which are taken shortly after the onset of the coma but may develop during the second day.

Insulin shock is very commonly associated with a diminution in the amplitude of the T wave and the T wave may become diphasic in Lead 1 or Lead 2. The administration of insulin in diabetic patients in sufficient doses to produce marked hypoglycemia has been observed to cause changes in the S-T segment and the T wave with great regularity. These changes were of such a character that the T wave and S-T segment came to lie

wave and a slight depression of the S T segment especially at the S T junction In patients with angina pectoris these changes are more marked and may lead to the appearance of an isoelectric T wave in Lead 1 or in Lead 2 or T_2 may become diphasic (—+) or inverted In one reported instance bundle branch block was induced temporarily Both in anginal patients and in normal individuals the effects last for from one to two hours

Nitroglycerin

The effect of nitroglycerin in patients showing inversion of the T wave has been studied by Evans and Hoyle Of 16 cases with inverted T in Lead 1 or in Leads 1 and 2 there was a return toward a more normal form of the T wave in 7 cases An inverted T sometimes became upright or less inverted whereas if Lead I showed the only inversion this T wave sometimes became upright With inversion of T in Leads 2 and 3 or in Lead 3 alone the effect of nitroglycerin was not noticeable in 5 cases out of 8 but in the other 3 the T wave became less negative or upright Nine cases were tested in which the T wave was upright in all three leads no change was noted in any of these Other observers have made occasional observations which are in general agreement with these These changes were considered to be related to the tachycardia induced by the nitroglycerin and as a rule lasted only from three to five minutes though in an occasional case the effect might last for twenty minutes

Nitroglycerin has also been observed to cause a prompt but transient return to normal of the S T displacement produced by epinephrine in animals It also tends to abolish the arrhythmia produced by toxic doses of epinephrine in animals

Atropine

The primary effect of atropine of course is to accelerate the heart rate and it produces little or no effect upon the form of the waves of the electrocardiogram When digitalis or nicotine is acting upon the heart atropine will have a synergistic effect enhancing the changes in the T wave characteristic of these drugs

opposite to the direction of the chief deflection of the QRS in the leads in which QRS showed its largest deflection The administration of glucose corrected these changes promptly With lesser doses of insulin similar changes are found but they do not reach the same degree

A low carbohydrate diet which produced hypoglycemia was found to produce changes of a similar type so it is likely that the changes are due to the low level of the blood sugar and not to the insulin With the low blood sugar level there is too little glucose available for the proper nourishment of the myocardium

When insulin shock is induced in patients who are not diabetics, as is done for instance in the treatment of schizophrenia similar changes appear in the electrocardiogram There is an increased auriculoventricular conduction an increased duration of QRS a depression of the S T segment and the T wave becomes smaller and eventually inverted The Q T interval is prolonged These are all considered to result from a slowing of the rate of conductivity in the muscle which probably results from a myocardial depression due to the lack of available blood sugar These changes all disappear after the shock has been recovered from but Hadorn observes that this is not necessarily a proof that no permanent damage has been done to the myocardium

Carbon Monoxide

Poisoning with carbon monoxide has been found to produce changes in the electrocardiogram That it does not always do so is indicated by the fact that even very severe cases may give normal records The most common abnormal features are (1) slight changes in the level and form of the S T segment either in the precordial leads or in the leads from the extremities or both (2) low amplitude or diphasic or inverted T 1 and (3) low voltage of QRS Less frequent changes are (4) inversion of T in Lead 2 (5) prolongation of the auriculoventricular conduction time and occasionally (6) intraventricular conduction defects These changes disappear as the asphyxia is recovered from and permanent changes are rarely if ever encountered Arrhythmias occasionally occur the most frequent being paroxysmal auricular fibrillation

Calcium

The most striking electrocardiographic feature associated with variations in blood calcium is the prolongation of the Q T interval found with hypocalcemia and its shortening with hypercalcemia The constant K is found to be greatly increased with hypocalcemia and to return to normal as the calcium in the blood returns to normal under treatment No other changes in the electrocardiogram have been associated with gradual changes in blood calcium After the intravenous injection of calcium gluconate there appears a temporary flattening of the T wave in Lead 2 with possibly a partial inversion which takes place within the first 30 seconds after the injection One minute after the injection the T wave has practically returned to normal Following this a marked bradycardia appears and simultaneously the T wave diminishes in height and may become partially inverted This bradycardia may last for twenty five or thirty minutes In a few patients there are no T wave changes and in about one half no changes in the P wave

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CHAPTER IX

THEORY OF THE ELECTROCARDIOGRAM RECOMMENDATIONS ON NOMENCLATURE

SINCE the electrocardiogram is due to the electrical disturbance associated with the contraction and relaxation of the cardiac muscle, it is necessary to review certain phases of electrophysiology if one is to have a proper understanding of the possibilities of the method. Recent work has shown the original negativity hypothesis of muscle excitation to be so defective that it seems hardly worthy of mention at present. The only belief which has survived from this theory is that the activity of muscle is associated with the production of a state of electrical negativity within the active muscle (Fig. 81 A).

Realizing that the older theory was not satisfactory, Lewis developed his theory of limited potential differences and supported it by observations upon the mammalian heart. According to this view, the muscle which is becoming active is relatively negative, and the as yet inactive muscle in union with it and immediately adjacent is relatively positive (Fig. 81 B). This will result in a difference of potential so placed in relation to the beginning of activity of the muscle that it moves forward with the crest of activity, always having the positive pole in advance of the negative. Since the current flows within the muscle from the negative to the positive, as within an electric battery, Lewis stated that the direction in which the current sets in the muscle will be the direction in which the excitation wave is at the moment travelling. He emphasized, however, that he did not believe that the direction of the current is dependent upon the direction of motion of the excitation, but that both are governed by one series of events in the muscle and in consequence are definitely associated.

As the contraction spreads the electrical disturbance progresses throughout the muscle the brief positive phase always in advance of the brief negative phase It is assumed that the spread

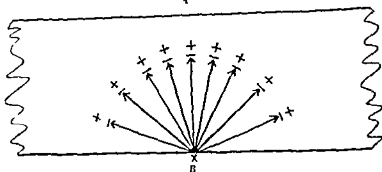


Fig. 79 Diagrammatic illustration according to the theory of limited potential differences of the spreading of the contraction through cardiac muscle from a point of stimulation at x on one surface of the tissue The arrows represent the radial spread from the point of stimulation The + and − characters indicate the positive and negative electrical phases which advance through the tissue at the forefront of the contraction wave. Points A and B represent the attachment of two electrodes at points remote from the muscle With such leading off points a potential difference would be indicated within the muscle having a direction from B toward A which is in the average direction of the spreading of the contraction

ing of the contraction is not confined in its progresses by the direction of the muscle fibers but that owing to the syncytial character of the heart muscle it spreads radially from the point of stimulation into all contiguous muscle fibers As the contraction process spreads in any one part of the heart muscle a difference of potential will be developed *within the muscle* having the same direction as the predominant spreading in that part This situation is shown diagrammatically in Figure 79

This theory was demonstrated in the mammalian heart by the experiment of Lewis described in his Mellon lecture of 1922 The heart of a dog was stimulated first on the epicardial surface of the lateral wall of the right ventricle and then by inserting an electrode into the cavity of this ventricle on the endocardial surface of the same wall Leading off contacts as for Lead I were placed on the sides of the chest wall The contraction wave in the first case spreads through the right ventricular wall from without inward which is from right to left The record obtained showed

an initial brief upward deflection indicating the production of a current within the heart from right to left when the contraction was spreading in this direction When the endocardial surface of the wall of the right ventricle was stimulated the contraction first spreads in a direction from within outward which is from left to right The record obtained under these circumstances showed an initial brief downward deflection indicating a current within the heart from left to right at this time Following these brief initial deflections the ventricular complex on each occasion showed a form characteristic of a premature contraction resulting from stimulating the right ventricle It was not materially different in the two cases

Although this relation between the direction of the initial spread of the contraction and the direction of the resulting electrical potential may be true for any section of the wall of the heart yet one must also consider the belief that a stimulus originating abnormally in the ventricular muscle will only spread radially through the muscle until it has effected a junction with the finer branches of the Purkinje tissue It will then spread through this tissue very quickly to relatively remote portions of the muscle where the stimulus will start other centers of contraction in the ventricular muscle By this means an orderly radial spreading through the ventricular myocardium is made impossible

Attention must also be called to recent anatomical observations that the Purkinje tissue penetrates into the ventricular wall almost as far as the pericardial surface and that in general it seems to follow the muscle bundles which have recently been re emphasized by Robb and Robb If the stimulus is transmitted *from the Purkinje fibers to muscle fibers at numerous points in* the ventricular wall this too will render impossible a radial spreading of the contraction such as Lewis considered likely Furthermore Lewis theory is difficult to apply to an explanation of the T wave

Craib proposed what he called a doublet theory* of the electrical manifestations of muscular activity (Fig 81 c) He believed that at the moment of activation doublets develop in the tissue

* A doublet consists of a positive and a negative pole of equal strength located very close together Any potential difference extending over a small space can be represented by a train of doublets

and progress as the contraction spreads with the positive pole always ahead of the negative pole These doublets of invasion as he called them remain for a brief period and as activity subsides

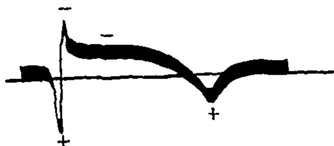


Fig 80 Deflection obtained by a direct lead from cardiac muscle Such a curve would be obtained with one electrode on the muscle lying in a large conducting medium and the other at a considerable distance from it (After Craib)

the doublets of retreat appear which consist of a negative pole preceding a positive pole This phase lasts somewhat longer than the stage of invasion Owing to this development of potentials if we lead off with one electrode from a point upon a muscle strip along which a contraction is spreading and if the other electrode is placed at a considerable distance in a uniform conducting medium we shall obtain a curve like that of Figure 80 He considered the electrocardiogram to be composed of a summation of many such curves from the individual muscle fibers of the heart as they become involved in contraction and relaxation

This theory made an important contribution to our understanding of the electrocardiogram and Wilson Macleod and Barker by means of a mathematical approach to the subject developed an amplification of Craib s theory which is more generally satisfactory (Fig 81 b) They were able to plot curves such as would be expected under certain experimental circumstances and to obtain very close agreement between these prediction curves and the actual electrograms obtained by experiment It was their conclusion that whatever may be the origin of the electrical currents associated with the excitation wave (depolarization) these currents are similar to those which would be produced if the crest of the excitation wave was immediately

preceded by a source (positive pole) and followed by a sink (negative pole) They suggested that the final deflection or T wave (repolarization) might be caused by a sink (—) followed by a

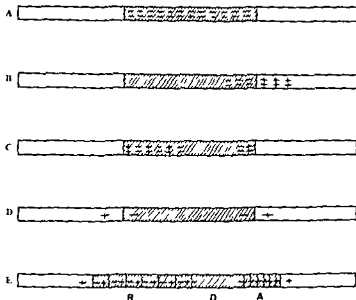


Fig 81 A diagrammatic representation of the various theories of the nature of the electrical manifestations of activity in cardiac muscle In each case the contraction is spreading from left to right The active region is shaded (A B C and D from Macleod A G *Am Heart J* 15 167 1938)

A The negativity hypothesis The entire mass of active muscle is represented as being negatively charged The inactive muscle is neutral

B The theory of limited potential differences (Lewis) A small region where the muscle has just become active is negative and the immediately adjacent resting muscle is positive

C The doublet theory (Craib) As muscle becomes active it becomes the seat of doublets whose positive element is toward the resting muscle When muscle regresses from the active state it gives rise to doublets of opposite polarity

D Bipolar theory (Wilson Macleod Barker) Ahead of the advancing boundary between resting and active muscle is a positive pole and behind it a negative pole Across the retreating boundary is a potential difference of reversed polarity but in this case the poles are farther apart

E Macleod's conception of the polarity changes accompanying the processes of accession (A) duration (D) and regression (R) of activity in a strip of cardiac muscle During the period of accession which is represented by section A of the strip there is a chain of doublets each having the positive pole ahead of the negative pole During the duration of activity in section D there is no change in potential and during the regression of activity there is a series of doublets each having its negative pole in advance of the positive There are the same number of doublets in the accession phase as in the regression phase so that those in the latter are farther apart that is weaker per unit of muscle length

source (+) but that the poles were farther apart in this case They were able to obtain some support for this theory from the

fact that on considering it in terms of the membrane theory of Bernstein they were able to reach a similar conclusion [illegible] further developed this theory and added a de[illegible] T [illegible]

the electrical processes accompanying the access[illegible] regression of activity in a strip of cardiac muscle During the stage of accession the advancing activity is accompanied by the production of a train of doublets each having the positive pole ahead of the negative the first positive pole being in the inactive muscle ahead of the onset of activity and each doublet in the train representing a greater stage of activity than its predecessor During the duration of activity there is no change in the electrical potential which remains negative During the regression of activity the tissue is the seat of a train of doublets each having the negative pole ahead of the positive one the last positive being in the inactive muscle behind the active tissue and each doublet representing a lesser stage of activity than its predecessor

The total sum of all the doublets of accession is equal to the sum of all the doublets of regression so that as seems more than likely if the stage of accession is shorter the doublets will be more concentrated in this region than in the one of regression which is of longer duration When recording the electrical activity within a strip of muscle accession and regression each will produce a diphasic curve the first phase of the curve of accession being + the second − The regression deflection will be a smaller and more prolonged diphasic curve which is also first + and then − This is illustrated in Figure 82 from Macleod which also shows the influence upon these curves of varying certain of the details of the muscle contraction by warming or cooling or by varying the relation of the leading off electrode to the long axis of the strip of muscle Based upon this conception Macleod devised a graphic method which demonstrated how the initial deflections (QRS) and the T deflection might arise They are the result of the overlapping of the curves of accession and regression which arise in the different parts of the myocardium

The time relations of the accession and regression deflections

preceded by a source (positive pole) and followed by a sink (negative pole) They suggested that the final deflection or T wave (repolarization) might be caused by a sink (—) followed by a

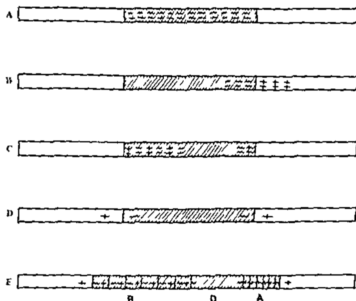


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source (+) but that the poles were farther apart in this case They were able to obtain some support for this theory from the

were found to vary with the duration of the active phase and the length of the muscle fiber

By heating the auricle of the frog it was possible to produce a record showing a separation of the deflections of accession from those of regression with only a small zone of overlapping so that it could be seen that they were both diphasic and oppositely directed (Fig 82 I) He developed a graphic method of analysis of the electrical events of contraction and relaxation which enabled him to predict the curve which would be obtained under various experimental conditions

Although the phase of regression is associated with a diphasic curve the first phase of this curve is ordinarily obscured because it coincides with the curve of accession The interval (S T segment) between the accession deflections (QRS) and the final phase (T) of the regression deflections will vary in its relation to the zero level lying upon it or above or below depending upon the relative duration of the phases of accession and regression and the duration of the full activity which intervenes between them

His experiments lead to the conclusions that the excitation process consists of (1) a brief stage during which activity is increasing (2) a very brief period of full activity and finally (3) a longer stage of decreasing activity

Wilson Macleod Barker and Johnston had previously discussed the T wave of the electrocardiogram and re emphasized Wilson and Herrmann s earlier statement that the order in which the ventricular muscle passes out of the refractory stage (contraction) is approximately the same as the order in which it enters into contraction Every change in the form of QRS produced by a change in the order of excitation was found to be accompanied by a corresponding change in the form of T opposite in direction to the change in QRS There were nevertheless certain factors which might change the form of T without causing changes in QRS such for example as cooling or warming the ventricular surface or the action of digitalis These factors probably act by affecting the duration of the contraction

They described a method of analysis of the electrocardiogram which makes it possible to determine the presence of influences affecting T but without coincident effect upon QRS After the

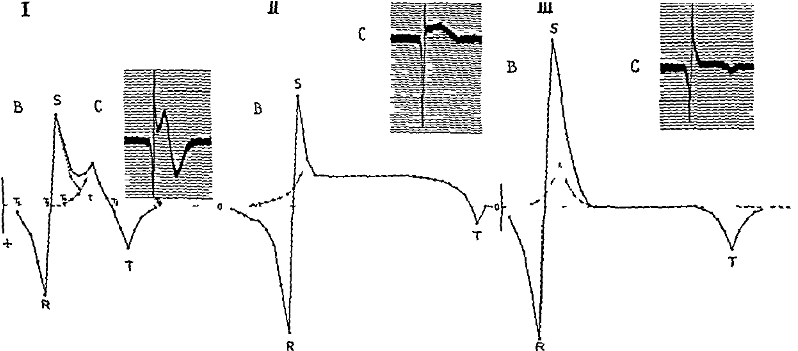


Fig. 82 Plotted theoretical curves *B* of the accession and regression of activity in a theoretical muscle segment to be compared with the *insets C* which are actual curves obtained by direct leads from a frog's auricle (These are slightly modified parts of Figures 9 10 and 11 from Macleod A C *Am Heart J* 15 165 1938)

I *B* The R and S peaks are the result of the accession effects the dotted line from S to T_1 is the plotted curve of the end of *the accession deflection* while the dotted line extending upward from T_1 represents the plotted curve of the onset of regression The solid line above the junction of T and T represents the summation of these two electrical effects I *C* *Actual electrogram from* the central region of a warmed frog's auricle

II *B* The plotted theoretical electrogram of a muscle strip the electrode being placed nearer to the end at which the impulse arrives first II C Actual electrogram from a point on a frog's auricle near the auriculoventricular *junction*

III *B* A similar plotting of the curves of accession and regression of activity in a short strip of muscle when the excitation process *is much longer than the muscle fiber* III C Electrogram from the central portion of a frog's auricle under ordinary experimental conditions

and size are determined by

ent parts of the ventricular muscle ... upon local factors which are independent of the order of excitation They might as has been said be the result of localized temperature differences affecting the myocardium or might be the result of a localized disturbance of the myocardium due to a localized disease process

When we consider the relationship of these electrical phenomena to the chemical and physical process of contraction we are confronted with a considerable deficiency in our knowledge The process of contraction has been divided theoretically into three parts The first is the phase of *excitation* which should be considered to comprise that preliminary activity which begins before and causes the actual shortening This phase is very brief The *contraction* proper is the second phase and comprises the physical shortening It is not known whether this begins before or after excitation is completed but it is probable that it begins either at the time of completion of excitation or very slightly before or after this time The more delicate and exact the method used for recording contraction the shorter has been found the interval between the onset of the electrical deflections and the onset of the contraction Complete contraction probably has a short duration and is followed by *relaxation* This seems to be definitely more gradual in its development than is the contraction

We do not know the time relations of the changes in potential to the three phases of contraction but we do know that the shortening begins very soon after the beginning of the electrical curve during the QRS deflections It continues until the latter part of the T wave about at its peak The initial brief electrical effects making up the QRS group occur during the excitation and the beginning of contraction while the later more prolonged potentials comprising the T wave occur at the same time as the chemical reactions during contraction and relaxation

We do not know the anatomical units in which this change in potential takes place but it is presumably some subdivision of

electrocardiogram was enlarged they measured with a planimeter the area included within the boundaries of the QRS deflection and the base line adding algebraically the upward (+) and downward (—) deflections Similarly the area of T was determined Having a value for the area of QRS and for T in each of the three leads the principles of Einthoven s triangle were applied and the angle α obtained for both the QRS and the T deflections Such an angle based on the area of the wave would indicate the direction of the average or *mean electrical axis* of QRS or of T instead of the electrical axis at a given instant, as is usually computed from individual deflections One may also compute the *manifest potential* of the *area* using the figures obtained for the area after the method suggested by Einthoven for computing the manifest potential (E) of individual deflections

The mean electrical axis of QRS would indicate the average direction in which the excitatory process spreads throughout the ventricular muscle and the mean electrical axis of T points exactly opposite to the average direction in which the recovery process spreads If therefore excitation and recovery take place in exactly the same order the angle between the electrical axis of QRS and that of T should measure 180° Likewise the area of T in each lead should be equal in magnitude but opposite in sign to the area of QRS so that the combined area of QRS T would be zero Differences between the area of QRS and the area of T may be expressed by algebraic summation thus obtaining the *manifest area of QRS T* Likewise the electrical axes of these two deflections may be added together giving the *electrical axis of QRS T* These two values—the manifest area and the electrical axis of QRS T—produce a vector* called by these authors the *ventricular gradient* This vector being the difference between QRS and T gives the direction and magnitude of the electrical forces resulting from a lack of uniformity in the duration of the excited state in different parts of the ventricles It expresses a difference between the potential of the QRS group and of the T wave and points in a direction from the region in which the average length of systole is greatest toward the region

* A vector is a force having a specified direction and magnitude

leads are obtained by an electrode somewhat remote from the heart muscle the negative deflection of these leads has been referred to by Wilson as an intrinsicoid deflection In Lewis work with experimental animals the wiring was so arranged that the negative intrinsic deflection was upward instead of downward This was also the case in the earlier work with human precordial leads It follows then that the peak of the R wave of precordial leads obtained with the present wiring indicates the beginning of the intrinsicoid deflection of the curve and therefore the time of the arrival of the contraction wave in the subpericardial fibers beneath the precordial electrode The downward deflection R S which follows is the intrinsicoid deflection and is due to the activity of the muscle fibers beneath this electrode

Influence of remote electrode It has been generally known that the precordial curves would vary somewhat depending upon the site of the remote electrode but the character and degree of the differences to be expected have not been well understood Certain authors have suggested the right arm or the leg or the left arm as the best site for the remote electrode but there has been much vagueness as to what is meant by the word best whether a large amplitude of the deflection or a greater tendency to show abnormalities

The supplementary report of the Committee of the American Heart Association on Standardization of Precordial Leads has indicated certain relations between the size of the deflections in the three limb leads that result from a given potential and the differences found in the corresponding deflection from a precordial point with different situations of the remote electrode The following numerical relations were pointed out referring in each case to the values in millimeters of any synchronous points in the leads mentioned such as is usually found in the peak of T

Synchronous points during the QRS group do not ordinarily coincide with the peaks of a wave in different leads

Lead 4 R = Lead 4 F + Lead 2
Lead 4 L = Lead 4 F + Lead 3
Lead 4 T = Lead 4 F + 1/3 (Lead 2 + Lead 3)

It is also true that Lead 4 R = Lead 4 L + Lead 1 By means of

the muscle fiber The P wave of the electrocardiogram is due to the summation of the brief initial effects produced by the accession deflections of the auricular muscle while the auricular T wave is produced by the final part of its recession deflections Similarly the QRS group results from the summation of the accession deflections of the ventricular muscle and the T wave from the final part of the recession phenomena

Direct and Semidirect Leads

The record obtained by a direct lead from the heart muscle placing one electrode directly upon the muscle and the other upon some remote region of the body is almost entirely the result of the potentials developed in the muscle beneath the point of contact with the electrode Potentials which are developed within the heart in regions remote from the point of contact contribute somewhat to such a record but the importance of their contribution diminishes rapidly with increasing remoteness from the immediate region of contact The record obtained by placing one electrode upon a cotton pad wet with saline which is lying upon the heart and the other upon some remote region differs but little and chiefly in magnitude from the record obtained when the electrode is in direct contact with the muscle Such a lead has been called a semidirect lead and it has been pointed out that leads obtained through the precordial tissues are also semidirect leads and when paired with an electrode upon some remote region such as the leg arm or back are almost entirely dependent for their form upon the potential beneath the precordial electrode

Wilson has pointed out that the development of the initial (+) deflection of precordial leads is associated with the approach of the contraction through the myocardium toward the subpericardial area beneath the electrode and that the peak of this initial deflection indicates the time of arrival of the contraction immediately beneath the electrode The deflection which follows this peak passes below the base line giving rise to a downward (—) deflection This negative deflection is analogous to what has been called the intrinsic deflection by Lewis and others following him when using direct leads from the heart muscle Since precordial

a regular progression of the electrical axis within the circle of Einthoven

INFLUENCE OF NEGATIVITY O[illegible] ELECTRODE IS PLACED UPON [illegible]

Angle of Vector	Deflection in Limb Leads 1	2	3	When Paired with a Precordial Electrode: The most + or le[illegible] − *deflection* will [illegible] obtained using limb of lowest [po]tential which will be					
90	0	4	4	L arm & R arm	4			L leg	0
1 0	−2	2	4	L. arm	4	R arm	2	L leg	0
136	−3	1	4	L arm	4	R. arm	1	L. leg	0
150	−4	0	4	L arm	4			R arm & L leg	0
104	−4	−1	3	L arm	4	L leg	1	R arm	0
±180	−4	−2	2	L arm	4	L leg	2	R arm	0
−150	−4	−4	0	L arm & L leg	4			R arm	0
−120	−2	−4	−?	L leg	4	L arm	2	R arm	0
−104	−1	−4	−3	L leg	4	L. arm	1	R arm	0
−90	0	−4	−4	L leg	4			R arm & L. arm	0
−60	2	−2	−4	L. leg	4	R arm	2	L. arm	0
−44	3	−1	−4	L. leg	4	R arm	3	L arm	0
−30	4	0	−4	L leg & R arm	4			L arm	0
0	4	2	−2	R arm	4	L. leg	2	L arm	0
30	4	4	0	R. arm	4			L leg & L arm	0
60	2	4	2	R arm	4	L arm	2	L leg	0
0	1	4	3	R arm	4	L. arm	3	L leg	0
)0	0	4	4	R arm & L arm	4			L leg	0

I[illegible] de to illust[ra]te the prediction [o]f qua[n]titat[ive] as w[e]ll as qualitat[ive] [d]iffer[e]nces arb[i]tr[a]ry [va]l[u]es have been g[iven] to th[e] l[i]mb lead deflections, [an]d an [ar]b[i]t[ra]ry v[a]lue [o]f [z]er[o] has be[en a]ss[u]med to the [s]mallest precordial d[e]flect[i]o[n] (right ha[n]d colum[n]) in ea[c]h [e]xamp[le]. The [value o]f the deflect[i]on [i]n the [o]ther tw[o] precordial l[e]ads has been calculated fr[o]m these fi[g]ures.

If the angle α of the deflections in this table is transferred to the diagram of Figure 21 it will be seen that there is a sector of 120 which is so related to each limb that when the angle lies within this sector a more positive or a less negative reflection will be obtained in the corresponding synchronous deflection of any precordial lead as follows

If the remote electrode of any precordial lead is placed upon the *right arm* the most positive (+) or least negative (—) potential

these formulas it is possible to determine the deflection in Lead 4 R if we have obtained Lead 2 and Lead 4 F and so on for any other deflections provided that synchronous points in the curve are considered It is rarely possible to make such conversions for the peaks of the QRS group because of the difficulty of determining *synchronous points during the course of these waves* The peaks of P and of T and the S T junction if the latter is of sufficient magnitude to be easily measurable usually can be treated without difficulty by these formulas If they fulfill Einthoven s formula in the limb leads they can be used for the above conversions

As an illustration of how these formulas may be used let us suppose that the deflections of the T wave are as follows

Lead 1 = 2 Lead 2 = 6 Lead 3 = 4 Lead 4 F = 5

Lead 4 L would then give a deflection of 9 (5 + 4) and Lead 4 R of 11 (5 + 6) If the T deflections were

Lead 1 = − 2 Lead 2 = 4 Lead 3 = 6 Lead 4 F = 5

Lead 4 L would be 11 (5 + 6) and Lead 4 R 9 (5 + 4) As another illustration

Lead 1 = 2 Lead 2 = −4 Lead 3 = −6 Lead 4 F = 5

In this case 4 L = −1 (5 + (−6)) and 4 R = 1 (5 + (−4)) It will be seen that the relation of the deflections in the limb leads has a determining influence upon whether 4 R 4 L or 4 F shall be the larger

Wolferth and Wood have indicated another method of approach to the relation between the deflections in the limb leads and the particular limb which might be expected to give a more positive (upward) a less positive or a more negative (downward) deflection when paired with any precordial point They constructed a table which indicated various relations of the size and direction of the deflections in the limb leads and in each case the particular limb which would be found to yield the most positive (least negative) or the most negative (least positive) deflection of the corresponding synchronous portion of the electrocardiogram when paired with a precordial point Table X is a modification and amplification of theirs including certain relationships of the deflections in the limb leads which are not found in their table and rearranging the order so that it conforms to

For routine use the leg occupies a satisfactory position in relation to most of the important deflections upon which we base many of our diagnoses and it is possibly for this reason that Geiger found that abnormalities are more frequently recorded with the electrode in this position than upon the right arm. In taking precordial leads we are desirous of obtaining a constant normal form with recognized normal variations during health and we also wish to obtain abnormal forms as frequently as possible in the presence of disease and to be able to attach definite significance to these variations. It will involve considerable further study to properly answer these requirements.

In order to eliminate the influence of the potential of the remote electrode Wilson has devised what he has called the *central terminal* which is constructed by connecting a wire from each of the three extremities to a single point each wire having in series a noninductive resistance of 5000 ohms. This central terminal produces a point which is so near to zero potential throughout the heart cycle that it may be so considered *without encountering serious error*. Leads from the precordium to this terminal give curves which are almost exact records of the potential variations of the surface of the chest beneath the electrode and represent as has been said semidirect leads from the portion of the heart beneath.

Precordial leads obtained by this technique seem at present to be as satisfactory as any of the others and it may be that the avoidance of the distortion due to the potential present at the limb electrode may prove to have practical advantages. This however is as yet uncertain for it is possible that the potential *of the limb* electrode may at times contribute a diagnostic feature to the record obtained from the precordium.

Indirect Leads

Remote leads from the limbs. The record obtained by leading off from two points at a relatively great *distance from* [illegible]

[illegible] are the conditions under which the limb leads are obtained. This summation of potentials holds for each of the

will be recorded when the corresponding part of the electrocardiogram in the limb leads has an angle lying between $-30°$ and $+90°$ It will be noted that the apex of this sector points toward the right shoulder

If the remote electrode is placed upon the *left arm* the most positive (+), or least negative (—) potential will be recorded from any precordial point when the corresponding part of the electrocardiogram in the limb leads has an angle α lying between $+90°$ and $-150°$ It will be noted that the apex of this sector points toward the left shoulder

If the remote electrode is placed upon the *left leg*, the most positive (+) or least negative (—) potential will be recorded from any precordial point when the corresponding part of the electrocardiogram in the limb leads has an angle α lying between $-150°$ and $-30°$ It will be noted that the apex of this sector points toward the left leg

Likewise the sectors which are associated with the most negative or least positive deflection in a precordial lead are as follows with the remote electrode on the right arm it is the sector between $+150°$ and $-90°$ with the remote electrode on the left arm it is the sector between $-90°$ and $+30°$ and with the remote electrode on the left leg it is the sector between $+30°$ and $+150°$ If these sectors are noted in Figure 21 it will be seen that each one includes the extremity concerned at its center

With these facts in mind we should be able to select a site for the remote electrode best calculated to reveal a positive or a negative deflection in a portion of the electrocardiogram which may be of particular importance at the moment If we have used a less favorable site for the electrode it is possible to derive from the deflection recorded the deflection which would have been obtained had the more favorable site been used This would be done by means of the formulas on page 323 It must be evident from these facts that there is no single site for the remote electrode which will always give the most positive deflection or the most negative deflection for any part of the electrocardiogram because the most favorable limb will depend upon the direction of the angle α of the vector of this portion of the curve as recorded in the limb leads

the opportunities for short circuiting afforded by the body tissue between the heart and the limbs This will vary from one individual to another and may vary at different times in the same individual It seems as though the amount of contact between the heart and the anterior chest wall had an important influence upon the size of the deflections obtained by the three leads for the recorded current (the voltage) is reduced by full inspiration Short-circuiting between the heart and the limbs may reduce the size of the deflections in the leads but it will not change the relative heights of the various waves for the short-circuiting affects each one alike

When the ventricles are contracting normally the speed with which the stimulus passes along the auriculoventricular and Purkinje system is so great that after a very small interval probably not more than 0 005 second the contraction will be spreading simultaneously in different regions of the ventricular muscle and in different directions in each region The electrocardiogram obtained by the three leads from the extremities does not record those potentials developed during the very earliest period of excitation of the ventricles because these potentials have not yet reached sufficient magnitude to be recorded by the remote leads The clinical electrocardiogram from its very beginning then must thus be composed of the resultant of numerous electrical forces of different sizes and directions arising from the different portions of the myocardium which are active at that time

The development of the P wave and of the QRS group depends upon the direction in which the *initial* potentials develop in the various parts of the auricular and of the ventricular muscle and this depends upon the order of activation of the muscle fibers and upon the relative size direction and position of the muscle masses If the order of activation is *constant* the potentials will be constant from beat to beat and the *form* of the curve unchanged This is the reason that the curves *obtained* from the same heart at different times are always the *same* unless the spreading of the contraction becomes abnormal *in* some way If the stimulus arises in an abnormal situation *an abnormal order* of activation will result and the curve will have *an abnormal form* If the presence of diseased tissue forces a normal stimulus

various units of time throughout the cycle of contraction. For this reason it is not proper to consider that the curves obtained by leads from paired extremities are comparable except in a general way with those obtained by leads from muscle strips or by direct leads from the heart or by semidirect leads from the precordium. They may have a QRS group and a T wave, but the electrical significance of these deflections is different in the semidirect and direct leads from their significance in the remote (limb) leads. In semidirect leads the muscle fibers nearest to the cardiac electrode have a much greater influence upon the curve than do those in more remote parts of the heart, while with the limb leads all muscle fibers are so nearly equidistant from both electrodes that all have approximately equal effects upon them.

With both types of leads, if different muscle groups give rise to simultaneous electrical potentials, these will be added to one another if in the same direction and will tend to neutralize one another if in opposite directions. If at an angle to one another they will be summated in so far as they are in line with the direction between the electrodes. A given potential has most effect if directly in line with the lead and least effect if perpendicular to it. Because of the predominant influence of the potentials beneath the cardiac electrode, these relationships are of little importance in semidirect leads. They are of great importance, however, in understanding the leads from the extremities. Records from the extremities will be a composite in size and direction of the various potentials present at each instant within the heart.

The electrocardiogram obtained by the three standard leads is not an adequate representation of potentials which occur in an anteroposterior direction. These leads best record those changes within the heart which occur in a vertical plane transverse to the body and passing through the shoulders and left hip. This is the plane represented in Figure 2. If potentials occur in another plane, they will be represented in the plane of the standard leads by a value equal to their projection upon this plane. The more nearly perpendicular they are to the plane of the leads, the less effect will they have upon it.

The record obtained by the three leads does not represent the full value of the electrical potentials within the heart because of

taneously Under these circumstances the contraction may be considered to spread through the ventricular walls from the inner to the outer surfaces in a radial manner as shown in Figure

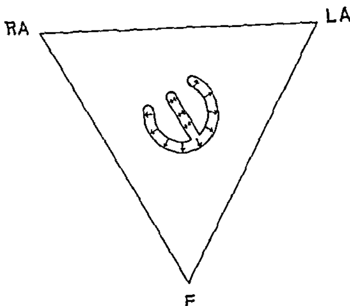


Fig 83 Wilson's diagram of the ventricular cavities The right and left ventricles and interventricular septum are readily recognizable The arrows indicate the direction of the spreading of the contraction through the ventricular muscle *from the endocardial to the epicardial* surface of the apex and lateral walls and from the endocardial surface of the septum on both sides (From Wilson F N Macleod A G and Barker P S *Am Heart J* 6 630 1931)

83 Opposite walls of the ventricles produce potentials which are opposite in direction and which therefore balance each other as long as each wall contains fibers which are entering into contraction When the spreading of the contraction reaches the epicardial surface the production of the initial potentials ceases in this direction so that if the opposite wall still contains fibers through which the contraction is spreading potentials will still be produced here in the direction of the spreading in this area He has further pointed out that owing to the gaps in the basal portion of each ventricle where the ventricular wall gives place to the valvular orifices the regions opposite to the valvular orifices will produce unopposed electrical potentials from the very be

to follow an abnormal path an abnormal curve will also appear Under either condition then, the form of the curve will differ from that associated with a normal contraction and normal muscle This explains the abnormal form of the complexes associated with cardiac arrhythmia and of those associated with myocardial disease

The QRS group The theories suggested by Wilson Macleod and Barker as to the production of potentials during the early part of the QRS group are the most satisfactory at present available for they are in harmony with most of the established observations from the clinic and the laboratory Mention should be made of certain observations which might tend to vitiate these theories though their importance has not yet been proved It has been shown by Abramson and by Roth that the ramifications of the Purkinje pathways run parallel to the muscle fibers in both ventricles and continue to within a very short distance from the pericardial surface Accordingly if the impulse is conducted to the ventricular muscle by the Purkinje fibers the contraction does not spread through the muscle tissue of the ventricles by a radial penetration starting from or near the endocardial surface Furthermore Wiggers has stated that all points on the pericardial surface of both ventricles except those opposite the edges of the interventricular septum are activated almost simultaneously The edges of the interventricular septum are activated earlier than other points on the ventricular surface Katz and his collaborators have insisted that the limb leads cannot represent equally the potentials produced in different parts of the heart because of the differences they have found in the electrical conductivity of the different tissues surrounding the heart They found the difference between the conductivity of the lungs and of the muscles to be of the order of 1 to 10 and believe that the regions of the heart in contact with lung tissue are but poorly represented in the limb leads as compared with the more favored regions in contact with the diaphragm and the paravertebral muscles and the anterior chest wall

Wilson and his associates have pointed out that the ventricles are irregularly shaped hollow cavities which are stimulated throughout the whole extent of their inner surfaces almost simul

the form of the curves of right or left bundle branch block may differ in different hearts because of the influence of hypertrophy or of areas of degeneration which might exist in one heart and not in another upon the fundamental or uncomplicated electrical production resulting from the lesion

In the normal electrocardiogram the simultaneous effects of the two ventricles are summated in such a way that opposing positive and negative forces neutralize each other and the resulting situation becomes so complex that it is not possible with our present knowledge to analyze it Lewis has shown that it is possible to combine mathematically the first portion of the electrical curve from the right ventricle with the first portion of the curve from the left ventricle and obtain a QRS group which is very much like that of the normal electrocardiogram of the dog from which the dextrocardiogram and the levocardiogram were obtained

The S T junction and T wave Much has been said about the approximate isoelectric level following the QRS group and before the peak of T It has been pointed out in discussing the clinical electrocardiogram that the S T junction may occasionally be isoelectric in one lead or in two but is very rarely isoelectric in all We realize now in view of what has been said about the development of the potential which goes to make up the T wave that the so called S T segment is only a portion of the T wave and that the T wave itself is only a portion of the potential produced by the regression of electrical activity T must represent in fact a summation of the last portion of the many diphasic curves which result from the regression of activity in the ventricular muscle It increases in voltage as more and more of the myocardial fibers cease their activity and finally reaches a peak This peak rapidly falls to zero as the remaining muscle fibers cease to be active Macleod has demonstrated that in muscle strips the deviation of the S T segment from the zero level is affected by the relation of the duration of contraction and the length of the muscle fiber Though this relationship may hold for experimental conditions yet it probably cannot be transferred directly to the heart contracting as a whole

Further information is needed concerning the relations of the

ginning of their activation For this reason the first electrical effects from each ventricle would have a direction determined by the spreading of the contraction in the apical portion of the muscle which lies opposite these valvular openings This direction will in general point away from the valve openings

For the human *right ventricle* these initial electrical effects have a direction which is *downward and toward the left* (+++), the tricuspid and pulmonic valves being placed to the right and above When the spreading of the contraction has pierced the wall of the right ventricle to the epicardial surface there will then be another area which has ceased to produce electrical effects This area is in the central region of the ventricle so that now the unopposed potentials in the interventricular septum opposite to this portion of the wall will produce forces pointing *to the left and upward* (++−) It will be noted that these two directions first downward and to the left (+++) then to the left and upward (++−) are represented in the electrical axes of the first portion of the curves associated with left bundle branch block the potential at this time arising exclusively from the right ventricle (Fig 86 D)

The human *left ventricle* has the valve openings in such a position that the apical muscle opposite to them is activated in a direction *downward and to the left* (+++) giving rise therefore to unopposed potentials in this direction during the first part of the QRS group As the contraction wave pierces the lateral wall in its apical portion where it is thinnest this region ceases to produce potentials and the portion of the septum opposite being now unopposed gives rise to forces directed *downward and to the right* (−++) in which direction the contraction wave is still spreading through it It will be seen that these directions first downward and to the left (+++) then downward and to the right (−++) correspond to the direction of the electrical axes during the early part of the curves associated with the right bundle branch block (Fig 86 E) the potential at this time arising only from the left ventricle A detailed comparison with the curves produced by ventricular premature beats is not proper for these curves must differ from the curves of bundle branch block because of the variable site of origin of the stimulus It is likely that

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The T wa nd the last deflection of the QRS group however are not al

obtained by

the following factors (1) The change in the position of the heart which occurs during systole tends to place the heart more in the long axis of the body during systole and to *rotate* it on its long axis so that a point anteriorly would move from right to left and from before backwards QRS is inscribed at least for the most part before this change of position has occurred and T almost entirely afterward (2) It is possible that certain factors may cause the relaxation of certain portions of the ventricular muscle to be slightly delayed or accelerated It is known that the application of cold to the surface of the ventricles will delay the relaxation and change the direction of the T wave and the taking of iced water into the stomach will do the same It is possible therefore that the proximity of the lungs to certain areas of cardiac muscle may make their temperature different from the temperature of other areas which are in contact with organs of a higher tempera ture such as the liver and that these temperature differences may cause a variable duration of the contraction of the subpericardial fibers thus changing the direction of T from that which it might otherwise have held

Explanation of difference of waves in the three leads It has been pointed out that the electrocardiogram is different when obtained by different leads This fact was at first a great stumbling block in the advance of our knowledge of the subject A wave which was upright in one lead was found much smaller in an other or even turned downward and it was not until Einthoven s mathematical explanation appeared in 1908 that this enigma was clearly understood Figure 11 contains the electrocardiograms of eight normal men as obtained by the three standard leads and it is evident at a glance that there may be as much variation be tween the three leads of one person as there is between the same leads of different persons

The explanation of this involves a good deal of mathematics but may be outlined by a series of diagrams which should not be difficult to follow Let us first examine Figure 84 which is like a record taken of the three leads at once by means of three gal

direction of the mean electrical axis of QRS and of T to variations in the relaxation of localized portions of the cardiac muscle The ventricular gradient as described by Wilson and his associates, is well calculated to bring out the influence of such differences

Confusion has resulted from attempts to correlate the T wave of the electrocardiogram with relaxation of the ventricular muscle Although its peak is reached at about the time that relaxation becomes dominant, yet the beginning of the development of this peak has taken place during the time when the contraction is in full force Whereas the QRS group occurs at the time of the invasion of the muscle by the contraction the T wave occurs during the persistence of the contraction and during its subsidence It is likely as has been said that the chemical changes producing the QRS group give rise to the shortening which constitutes the contraction The T wave starts so early that it could scarcely be due to the relaxation of even small numbers of fibers It is important to realize that the electrical curve probably is more closely associated with the chemistry of the contraction process as a whole than with the physical processes of shortening (contraction) and lengthening (relaxation) of the fibers We must consider the likelihood that the T wave is not at all related to relaxation but merely to an anabolic change which balances the katabolism which gave rise to the QRS group and the contraction Relaxation may not be related to chemical or electrical changes It may be purely physical

In the normal heart muscle the contraction process will tend to last the same length of time in all muscle fibers so that the potential producing T will appear first in those parts which were first invaded by the contraction process and last in those parts last affected For this reason the direction of the T wave of the electrocardiogram should be more closely related to the direction of the final deflections of the QRS group than to its earlier ones and should tend to be opposite to them

In the electrocardiogram obtained by the standard leads the final components are summated just as are the initial ones but owing to the slower development of the potential of regression the successive peaks of the initial phases are not reduplicated

Einthoven demonstrated that the three leads may be considered to form an equilateral triangle standing on one of its angles and having the heart at its approximate center as shown in *Figure 2* The leads do not form an exact equilateral triangle but the error from considering that they do so is a negligible one Let the arrow through the heart in *Figure 2 represent a potential* within the heart its direction representing the direction of the potential and its size the size of the potential *When such* a potential is recorded by a galvanometer by leads placed about it forming a triangle as do the three standard *leads* the record by each lead will bear a *certain proportional relation to the actual potential* The proportion recorded by *each* lead can be determined by projecting the arrow representing the potential perpendicularly upon that side of the triangle which represents the lead This projection is shown by the dotted perpendiculars drawn from each end of the arrow to the sides of the triangle and it can be seen that the length of the projection is different *in each* lead Note also that if the potential within the heart is perpendicular to one of the leads its projection upon that lead will be zero and upon the other two leads equal

Since the three leads form a triangle about the heart an electrical potential within the heart *could not* possibly be represented in *the same* way upon all of them It is as if we should look at an approaching railroad train from three different directions *each direction* being represented by the position of one of the sides of *the triangle* If we look straight at the end of the train it will appear not to have any direction (*i e* to left or right) nor any length From any other two *directions* however its length and the direction of its motion will be evident If instead of being straight in front or behind the train we are even a little to one side then it will appear to move toward the right or the left as the case may be and its length will appear *greater* and greater the more it deviates from the end on *direction Only* when it is running at right angles to our line of vision can we appreciate its true length This is the *condition* which corresponds to the electrical force being parallel to the line of a lead

As has been mentioned already the amount actually recorded in the leads will be less than the potential within the heart on

vanometers recording upon the same photographic plate and within the same network of time lines The speed of the photographic plate is much magnified as is also the distance between

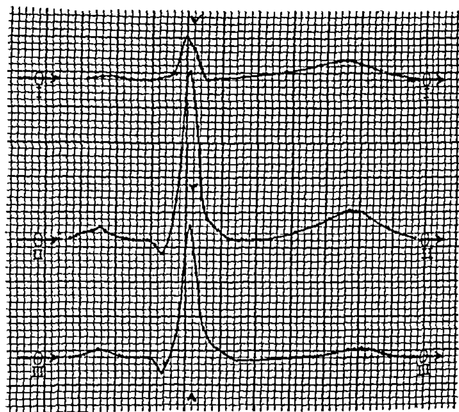


Fig 84 To show the electrocardiogram by the three limb leads in the same system of time lines as if taken synchronously (From Lahr *Heart* 1 162 1910) The space between horizontal lines represents as usual 0 1 millivolt but the space between vertical lines represents 0 01 second so that the curves are spread out horizontally

the horizontal lines so as to facilitate careful measurement A record of the electrical potential within the heart at each instant thus falls upon the same vertical time line in each of the three leads On measuring the height upon the same time line in each lead it will be found that the three leads never have the same excursion at the same instant though any two leads may be alike The electrocardiogram is obviously composed of a succession of brief electrical effects such as the ones we have measured Let us now consider why it is that the same identical electrical potential within the heart is recorded differently in different leads

Figure 83 A shows a potential within the heart whose direction is more *toward the patient's* right than that shown in Figure 2 This would cause a downward deflection in Lead 1 and an up-

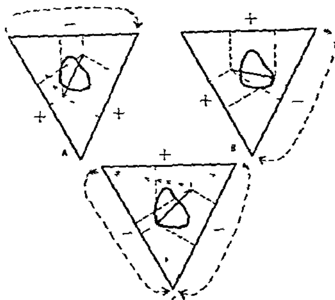


Fig 83. Representing the direction of the potentials within the heart which will produce three different combinations of deflections in the three leads. The triangle heart and arrows have the same significance as in Figure 2

A = direction to produce downward (−) deflections in Lead 1 and upward (+) deflections in Leads 2 and 3

B = direction to produce downward (−) deflections in Lead 3 and upward (+) in Leads 1 and 2.

C = direction to produce upward (+) deflections in Lead 1 but downward (−) in Leads 2 and 3

ward deflection in Leads 2 and 3 (−++) The direction of the deflection in Lead 1 is changed because the direction of the potential within the heart causes the current to flow through the galvanometer from right arm to left arm instead of the reverse Figure 83 B shows a potential within the heart which is directed more toward the patient's left than that shown in Figure 2 The deflections will be upward in Leads 1 and 2 but downward in Lead 3 (++−) The Lead 3 deflection is downward because the flow through the galvanometer is from arm to leg instead of from leg to arm as in Figure 2 Figure 83 C shows a potential within the

account of the short circuiting within the body, but the proportional representation in the three leads is believed to be correctly maintained. To represent the effect of short circuiting the dotted lines from each end of the arrow to the sides of the triangle of the figure should be converging instead of parallel as they approach the sides.

We obtain from an experiment by Fahr an idea of the relation of the size of the potential within the heart to the potential recorded in the leads. He placed two electrodes in the heart of a cadaver and caused a potential difference of 0.2 volt between them, meanwhile taking a record of the resulting deflections by the three leads in the usual way. His records showed the following values:

Lead 1 = 1.0 millivolt
Lead 2 = 4.6 millivolt
Lead 3 = 3.6 millivolt

a proportion of 200:1, 200:4.6 and 200:3.6 for the three leads, which would indicate that in the largest lead we may record about 2.3 per cent of the heart's potential.

The flow of this current from the heart through the limbs to the extremities is indicated in Figure 2 by the line of arrowheads for Leads 1 and 2, and the direction of flow through the galvanometer is represented outside the triangle by the dotted arrows for all leads. The feathered end of the arrow is toward the limb by which the current leaves the body and the head toward the limb by which it reenters. A current like that represented would cause an upward deflection of the record in each of the three leads because of the standard method of connecting the wires from the limbs to the galvanometer.* Though free use has been made of the word current in this explanation, it must be realized that the potential differences within the heart produce very little current, that is, the amperage would be very low. When two leads are taken simultaneously with two string galvanometers the amount of current passing through one galvanometer is sufficient to definitely diminish the amount of current available for the other.

* The wires from the limbs are connected to the galvanometer so that a current passing through it from left arm to right arm or from the leg to either arm will cause an upward deflection in the record.

or pericardial fluid There is at present little definite knowledge on these points

The angle alpha In designating the direction of potentials within the heart Einthoven considered all directions in relation to the horizontal which is parallel to Lead I The horizontal toward the patient's left he called 0° the 180° above this were given negative (−) values the 180° below were considered positive (+) values Thus 180° is horizontally to the patient's right +30° is downward to the left and −150° is upward toward the right exactly opposite to +30

The diagram of Figure 21 (page 46) represents this idea The circle is drawn in such a way as to include within it the triangle representing the three limb leads of Einthoven It is divided into segments one side of each segment being a line drawn through the center of the circle and perpendicular to one of the sides of the triangle the other side of the segment being a line from the center of the circle to an apex of the triangle so that each segment includes an angle of 60 Within each segment the plus or minus sign opposite the values shown indicates the direction of the deflection which would be produced in the respective leads by potential differences within the heart having a direction parallel to any of the radii within this segment If the potential difference has a direction parallel to the dividing line between two segments it will be perpendicular to the direction of one of the leads and the deflection will be zero in this lead If the direction is nearly parallel to a dividing line (perpendicular to a lead) then there will be but a small value in the lead an upward (+) deflection if it is on one side and a downward (−) deflection if on the other side of the perpendicular The analogy to the train is to be recalled for the potential causes no deflection in the lead toward which it is exactly directed just as the train has no length when viewed end on

We see that variations in the direction of the potential within the heart will affect the relative size of the excursions in the three leads and will also determine their direction so that they may be upward or downward Variations in the size of the potential within the heart will cause changes in the size of the waves of all

heart which causes a downward deflection in both Leads 2 and 3 while that in Lead 1 is upward (+——) This current is directed still farther toward the patient s left

Einthoven s law of the values in the three leads Einthoven showed that there is a mathematical relation between the size and the direction of the movements in the three leads resulting from any potential within the heart If the deflections in the leads are measured at simultaneous portions of the curve it will be found that

Value Lead 1 + value Lead 3 = value Lead 2

This is true for positive values (upward deflections) negative values (downward deflections) and for combinations of positive and negative values The figures obtained with an artificial potential within the heart have been found to agree with this formula and if it be applied to measurements upon the same time lines in Figure 81 it will be found to hold good with a very slight error If we can decide upon the parts of any record which are simultaneous in the three leads we shall find that they fulfil this formula

If we know the size and direction of the deflection in each of the three leads we can reverse the process of Figure 2 and construct the size and direction of the hypothetical electrical force within the triangle which would produce deflections in the leads like those we have measured This hypothetical force was called by Einthoven the *manifest potential* (Em)—as opposed to the *evident potential* (e) which is that recorded in the leads The manifest potential is a hypothetical value and not a potential which truly exists anywhere but still we can determine its value and compare its size and direction on different occasions because it bears a certain proportional relation to that true potential within the heart which we are unable to measure The ratio between the true potential and the manifest potential may not be a constant one It is possible even likely that there may be different degrees of short circuiting within the body of different individuals and even in the same individual this may be varied by certain variable conditions outside of the heart such as emphysema

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decreases in size the direction of the vectors meanwhile varying irregularly in a clockwise direction During the T wave the potential rises and falls but there is no tendency toward a regular variation of the direction of the current

This increase and decrease of the size of the vectors of P QRS and T is found in all electrocardiograms It is usually a regularly progressive variation but at times may be irregular The variation in the direction of successive vectors differs also in different records This is particularly the case with the vectors of the QRS group Sometimes these will be progressively rotated in one direction sometimes progressively in the other and sometimes they will vary quite irregularly the angle pointing now one way and now another without any predominant trend

Records like those of Figure 16 usually show a regular rotation of the vectors of the QRS group in a clockwise direction starting at about − 40 and ending at + 120° Records like those of Figure 17 usually show a regular rotation of the vectors of QRS in a counterclockwise direction starting at about + 90° and ending at about − 20° or − 60° Records A and B of Figure 11 would probably show a rotation with the clock and record C against the clock but it is rare for normal records to show such a smooth uninterrupted sweep of the vectors as do the records with right or left axis deviation of QRS

Mann has devised a mathematical method for constructing a graph which he calls the *monocardiogram* This graph takes its form according to the successive variations in the size and direction of the vectors of the electrocardiogram It introduces nothing which is not already present in the record obtained by the three leads but it makes for a more ready appreciation of the variations in the size and direction of successive vectors It seems likely that along this or a similar line much may be learned of the normal and abnormal electrocardiogram which could not be obtained by simple inspection of the record by the three leads

Wilson and Johnston have constructed an instrument capable of recording such a curve from the usual limb leads They have named their records *vectorcardiograms* to emphasize that their inscription follows the path traveled by a line joining the tips of the successive vectors of the cardiac potentials This might be

the leads proportionately, but will not change the direction in any lead

The electrocardiogram a series of vectors The electrocardiogram is the record of an electrical potential which is constantly varying in size and direction It may be considered as the record of a succession of different potentials each one differing slightly from the preceding one, just as a motion picture is a succession of different views of a moving object each separate view being slightly different from the others A force having both size and direction is called a *vector* and it may be said that the electrocardiogram is produced by a series of electrical vectors

It must be clearly understood that the electrocardiogram as obtained by the three standard leads does not record actual potential differences which exist in the heart but that the record at any given point of its course expresses the net result of the many potentials which exist simultaneously in the different parts of the heart Many of these potentials are undoubtedly in opposing directions so that their values tend to neutralize each other partially if not wholly The vector recorded at a given instant may not have the direction of any of the electrical forces within the heart that go to produce it

We can determine these vectors from the record by the mathematical process explained in detail in the appendix or by the use of the geometric diagrams of Figures 101 or 102 For example on the time line designated by the arrow in Figure 84

e 1 = 4 5 mm e 2 = 21 mm e 3 = 16 5 mm

Using Table XIII in the Appendix we find that these deflections must have been due to a potential at + 78° with a manifest value of 2 2 millivolts The vector for the next time line

e 1 = 1 7 mm , e 2 = 9 mm e 3 = 7 3 mm

is 0 95 millivolts at + 79° and so the process may be carried on throughout the whole of the electrocardiogram

During the P wave of Figure 84 the potential first increases and then decreases in height and the angle of the vectors gradually changes from about + 90° to about + 30° so that the vectors rotate in a counterclockwise direction toward the patient s left During the QRS group the potential first increases and then

decreases in size the direction of the vectors meanwhile varying irregularly in a clockwise direction During the T wave the potential rises and falls but there is no tendency toward a regular variation of the direction of the current

This increase and decrease of the size of the vectors of P QRS and T is found in all electrocardiograms It is usually a regularly progressive variation but at times may be irregular The variation in the *direction* of successive vectors differs also in different records This is particularly the case with the vectors of the *QRS* group Sometimes these will be progressively rotated in one direction sometimes progressively in the other and sometimes they will vary quite irregularly the angle pointing now one way and now another without any predominant trend

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done in Figure 86 L and D and would enclose in each case an irregularly shaped loop similar to the QRS loop of the vectorcardiogram.

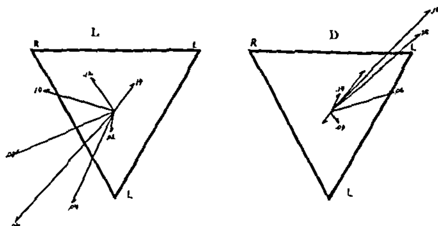


Fig. 86. Direction and size of the vectors of the record of Figure 87. The triangles represent the leads as in Figure 2. The direction of the arrows represents the direction of the vectors. The length of the arrows represents the size of the vector (Em). The figures indicate the time in seconds from the beginning of the QRS group. These diagrams are constructed from the figures in Table XI.

Lewis has shown in the dog that when the branch of the auriculoventricular bundle passing to one ventricle is cut across the electrocardiogram for the first 0.04 or 0.05 second will represent only the activity of the ventricle whose bundle branch is intact. After this interval the other ventricle also becomes active receiving its stimulus through the muscle of the interventricular septum from the first one. He applied the terms dextrocardiogram and levocardiogram to the curves resulting from the exclusive activity of the right ventricle and left ventricle respectively. The dog's dextrocardiogram shows upward initial deflections in all three leads. The vectors steadily increase in size and rotate uniformly in a clockwise direction as do those in Figure 86 L. The dog's levocardiogram may be either of two general types, one showing chiefly downward initial deflections in all three leads and the other which Lewis calls "discordant" giving upward deflections in Lead I and downward deflections in Leads 2 and 3. Either type of levocardiogram was shown to consist of vectors which steadily increase in size and rotate uniformly in a

counterclockwise direction as do those in Figure 86 D He found that by combining mathematically the values of the vectors of the dextrocardiogram and levocardiogram from the same animal he

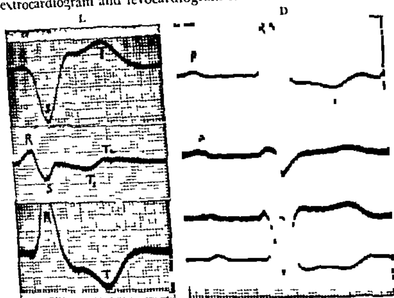


Fig 87 The three leads are mounted as if taken synchronously The speed of the photographic plate was such that each of the time lines represents 0 01 second
L A premature beat arising in the left ventricle
D One of the usual ventricular complexes of this patient indicating left bundle branch block with slightly prolonged P R interval (0 23 sec) The record of Lead 3 has a superimposed record by Lead 1 which can be seen to be like Lead 1 of this illustration

was able to reproduce a QRS group practically identical with the normal curve of the dog in question

Human dextrocardiograms and levocardiograms as obtained from records indicating a lesion of one bundle branch show considerable differences from those of the dog but also show a progressive rotation of their vectors though in a different direction from those of the dog In view of the recent observations mentioned in the discussion of the localization of bundle branch block (page 117) it seems that the human dextrocardiogram is characterized by initial deflections which are at first either −++ +++ or perhaps rarely ++− and with the later vectors rotating in a counterclockwise direction while the levocardio

done in Figure 86 L and D and would enclose in each case an irregularly shaped loop similar to the QRS loop of the vector cardiogram

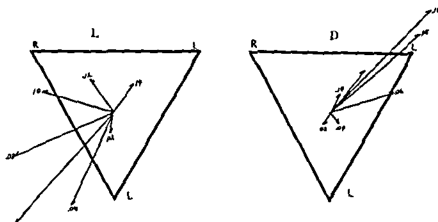
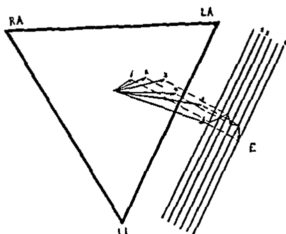


Fig 86 Direction and size of the vectors of the records of Figure 87 The triangles represent the leads as in Figure 2 The direction of the arrows represents the direction of the vectors The length of the arrows represents the size of the vector (Em) The figures indicate the time in seconds from the beginning of the QRS group These diagrams are constructed from the figures in Table XI

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the dextrocardiogram and levocardiogram are completed the progressive rotation of successive vectors continues though at this time both ventricles are contributing potentials Compare also



notch would be in Lead 3

The numbered radii are the vectors at successive units of time. The curve is replotted from the vectors upon the series of lines at the side of the triangle each of these corresponding to the similarly numbered vector This replotted curve *E* would be the electrocardiogram by Lead 3 if the successive potential differences within the heart were as represented by the vectors

the direction of the vectors during the early part of QRS with the explanation of the production of these vectors on page 332 It may be mentioned that Lewis has published an analysis of curves obtained from a patient who showed what we now consider to be a lesion of the left bundle branch and in this curve the direction of the vectors during the dextrocardiogram varied from 120° at first quite uniformly in a counterclockwise direction reaching 16° by 0 05 second after the beginning of the QRS group Likewise in a curve indicating a lesion of the right bundle branch he found the vector at the beginning of the QRS group in the resulting levocardiogram at 60° and then rotating quite uniformly in a clockwise direction to reach 170° by 0 05 second after the beginning of QRS Wilson has found a similar rotation of the vectors in records of these two types

gram has its initial deflections either +++ or ++− and later vectors rotating in a clockwise direction

Figure 87 D is a human electrocardiogram indicating a left bundle branch lesion the first 0 06 second of which probably may be considered to represent the human dextrocardiogram Figure 87 L is a record of a premature beat starting in the left ventricle of the same patient the first 0 06 second of which probably may be considered to represent the human levocardiogram though less exactly than would a bundle branch block complex for the reasons given on page 115

TABLE XI

VECTORS DERIVED FROM CURVES OF FIGURE 87

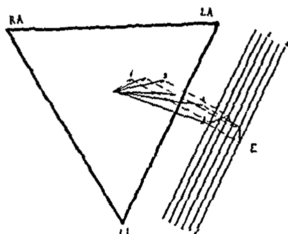
Time From Beginning of QRS	87L		87D	
	Angle of Vector	Size of Vector (Em)	Angle of Vector	Size of Vector (Em)
02	100	3 0	120	2 0
04	114	15 0	52	2 0
06	132 ?	22 0?	− 18	10 0
08	156	16 0	− 42	18 0
10	−164	1[illegible] 0	− 46	22 0
12	−127	6 0	− 52°	8 0
14	− 56	5 0	− 66°	3 0

Table XI illustrates the angle and the value of the vectors at successive intervals of *0 02 second throughout the QRS group* of these two curves Synchronous points of time in the three leads were determined by taking Lead 1 and Lead 2 simultaneously *upon the same photographic plate by means of two galvanom*eters and then Lead 1 and Lead 3 simultaneously *

Figure 86 was constructed from the figures in this table In Figure 86 D the dextrocardiogram starts at −++ and shows a counterclockwise rotation of the vectors while in 86 L the levocardiogram also starts at −++ but shows clockwise rotation It is interesting and noteworthy that even after 0 06 second when

* These records were made in the laboratory of the Department of Physiology of the College of Physicians and Surgeons Columbia University under the direction of Prof H B Williams

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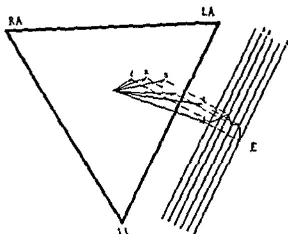


Fig 88 Showing how notches may arise in one lead when the successive vectors rotate toward the perpendicular faster than they increase in size or vice versa away from the perpendicular faster than they decrease in size In this case the notch would be in Lead 3

The numbered radii are the vectors at successive units of time *The curve is* replotted from the vectors upon the series of lines at the *side of the triangle* each of these corresponding to the similarly numbered *vector* This replotted curve E would be the electrocardiogram by Lead 3 if the successive potential differences within the heart were as represented by *the vectors*

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The electrical basis of notching If the vectors of an electrocardiogram rotate irregularly by jerks while approaching the direction perpendicular to a lead and their height increases

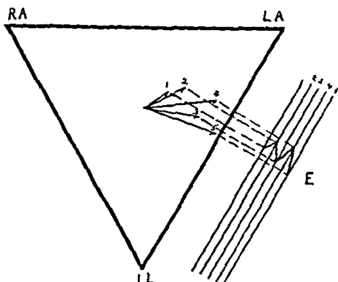


Fig 89 Showing how notches may arise when the successive vectors do not rotate regularly even though they increase or decrease regularly in size The production of the notching is shown for Lead 3 but Leads 1 and 2 would also be notched with these vectors

regularly at the same time the resulting wave will show a more or less pronounced notching or a thickening or slurring of the ascending or descending line Figure 88 shows diagrammatically how an irregular rate of rotation of the successive vectors can cause notching or slurring of the descending limb of R in Lead 3 which is the lead of small excursion for the QRS group represented Notching or slurring caused in this way is found either in a lead of small relative excursion or near the baseline of one of large relative excursion If the vectors rotate irregularly while leaving the direction perpendicular to a lead their height decreasing the while then the situation is the same as that in Figure 88 except that the timing is reversed

A change in the direction of rotation of successive vectors whether from clockwise to counterclockwise or vice versa may cause notching or slurring near the peak in the lead of small excursion and perhaps also near the baseline in a large lead This situation is shown in Figure 89

Notching or slurring may appear even though the rotation of the vectors is regular and constant if the size of successive vectors varies irregularly. Figure 90 shows this condition. Notching or

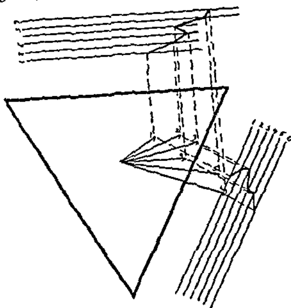


Fig 90 Showing how notches may arise when the successive vectors do not vary regularly in size even though the rotation is regularly in one or another direction. Here the notching is in Leads 1 and 3 but most in Lead 1 which is more nearly parallel to the vectors. Lead 2 would not show notching but would be slurred.

slurring from this cause will be more evident in the lead of large relative excursion, which is the one more nearly parallel to the direction of the vectors. This is in contrast to the notching or slurring produced by irregular rotation of the vectors, which shows most plainly in leads of small relative excursion or near the baseline of larger leads.

These mechanisms of notching apply either to P or to the QRS group. Such variations in the development of the potential depend upon the manner in which the contraction spreads over the muscle. The notching of QRS which appears in a small lead or near the baseline of a large lead is due to irregularities in the rotation of the vectors or to lack of coincidence in the rates of rotation and of increase in size. This does not usually indicate

an abnormality of the muscle The notching which appears in the QRS group in large leads usually near the peak of the wave and usually appearing in two or three leads is not found in records from normal hearts It is due to irregular variations in the size of the successive vectors as in Figure 90 and must be considered to indicate an abnormality of the spreading of the contraction in the muscle of the ventricles Notching near the peak of the P wave however is commonly found in normal records in two or more leads and is due to irregular variations in the development of the normal auricular current

When the variation in the size of the successive vectors is irregular coincident irregularity of rotation of the angle may increase the notching or may partly counterbalance it The two mechanisms may work with each other or against each other

Nomenclature and Description of the Electrocardiogram

There is at present a regrettable confusion in the application of Einthoven s terminology of the peaks of the electrocardiogram (Hurxthal Shookoff and Douglas Vega and Quero) This is especially true of the QRS group and to a lesser extent of the T wave It is unfortunate that our terminology should be indefinite for the methods most used for describing and comparing the curves involve the measurement of the height of the peaks It is necessary therefore that the peaks be similarly named by all who are studying them Because of this confusion the author has recently suggested a uniform nomenclature which can be easily applied and which may offer a solution of the various difficulties that have been encountered These suggestions and the considerations which gave rise to them are as follows

Einthoven at first designated the usual large upward deflection of the QRS group as R and applied the name Q to the downward deflection which often preceded it and S to the downward deflection which often followed it When he came to compare records obtained by three leads from the same individual he encountered difficulties in applying this terminology to the peaks appearing in different leads It was apparently his idea at this time that R should be the name of the most prominent peak of each lead and that the terms Q and S respectively should be applied

to the portions of the QRS group preceding and following this peak He referred repeatedly to the most prominent peak *in* Lead 1 or Lead 3 as an R wave even when these peaks were directed downward and occasionally referred to an upward Q or S wave In Westerns uber etc in 1908* there is a QRS group like that of the end of Figure 18 and he has labeled the upward peak of Lead 3 as Q and the downward one R In this same article he suggested that the formula Lead 2 — Lead 1 = Lead 3 might be used for the identification and naming of the corresponding peaks in the different leads He stated that although this method might appear simple enough yet difficulties would arise in applying it especially because the waves indicated by the same letters often fail to occupy identical phases of the heart cycle

Quite evidently he *recognized* at this time the fallacy of considering that similarly *named* peaks in different leads would always be due to the same production of potential within the heart Possibly it was the further development of this thought which led him in 1916 in referring to Lead 3 of a record with a typical left axis deviation of QRS such as Figure 17 B to say that since there is no basis to consider this large negative peak (in Lead 3) as either R or S we encounter a real difficulty in searching for the similarly named peak in the other leads We avoid this difficulty if we consider the QRS group as a whole and take the highest or lowest point of the group in the three leads as the object of our measurement The measurement referred to was to be used for determining the electrical axis of the most prominent peak of the QRS group In his later writings he showed little tendency to name the individual *peaks* Among the illustrations of his Harvey Lecture in 1924 he showed Lead 1 and Lead 3 of a record quite similar to that of Figure 17 B and marked the QRS group of Lead 3 simply as QRS in spite of the fact that naming the peaks would not have been difficult in this case if his original procedure of calling the largest deflection R had been followed

Einthoven's terminology as he first used it seemed to imply that peaks of the same name in different leads should represent the same current within the heart As he himself pointed out

* Figure 41 page 81

an abnormality of the muscle The notching which appears in the QRS group in large leads usually near the peak of the wave and usually appearing in two or three leads is not found in records from normal hearts It is due to irregular variations in the size of the successive vectors, as in Figure 90, and must be considered to indicate an abnormality of the spreading of the contraction in the muscle of the ventricles Notching near the peak of the P wave however is commonly found in normal records in two or more leads and is due to irregular variations in the development of the normal auricular current

When the variation in the size of the successive vectors is irregular coincident irregularity of rotation of the angle may increase the notching or may partly counterbalance it The two mechanisms may work with each other or against each other

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S to a peak in Lead 1 resulting from potentials which are a part of R in Lead 3 and the name Q in Leads 2 and 3 to peaks resulting from potentials which are a part of R in Lead 1 It also fails to indicate by name the deflections in Leads 2 and 3 caused by the potential producing S_1 or to indicate in Lead 1 the potential producing the deflections Q and Q_3 In each case the value in question is represented in the ascending or descending limb of the R wave It would have been evident that this was the case if Einthoven s formula of lead values had been applied to the usual clinical electrocardiogram of this patient The negative value of Q_3 must have a corresponding positive value in Lead 1 of at least the difference between Q_3 and Q and the negative value of S_1 must have a corresponding positive value in Lead 3 of at least the difference between S_1 and S Thus the manner of development of the heart s potential often can be roughly deter [illegible] on of Einthoven s formula to the peaks [illegible]

it On the contrary if they be taken too literally the [illegible] that like names in different leads represent like potentials will actually become misleading It is possibly for this reason that he came to avoid naming the individual peaks and to speak of the QRS group as having its chief deflection in one or another direction

Lewis and Gilder in 1912 published a series of measurements of human electrocardiograms and in this report as well as in his other writings Lewis deviated somewhat from Einthoven s usage in the method of lettering the peaks of QRS He indicated only downward peaks by the letters Q and S and only upward peaks by the letter R With this method of naming the peaks the upward deflection of Lead 1 might be called R while a corresponding synchronous downward deflection in another lead due to the same potential might be called either Q or S depending upon whether it preceded or followed the upward deflection of that lead which would be called R When the upward deflection was notched or when there were two upward peaks Lewis referred to these deflections as a notched R wave

Lewis method of lettering the peaks failed as did Einthoven s

however, this implication is not justified for the different peaks of the QRS group in the three leads rarely occur at exactly the same instant during the cardiac contraction and sometimes are

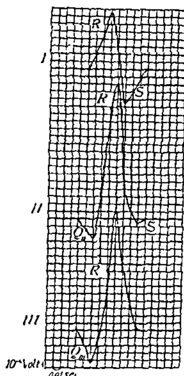


Fig. 91 Construction of the three leads of an electrocardiogram within the same system of time lines (From *Arch f d ges Physiol* 150, 91, 1913)

markedly separated in time The similar name does not indicate the same action current of the heart This feature may be demonstrated clearly if two or more leads are taken simultaneously Figure 91 which is reproduced from Einthoven affords an excellent example of how peaks named alike by Einthoven's method may nevertheless fail to represent synchronous portions of the QRS group The peaks of Q and Q_3 occur during the ascending limb of R_1 The peak of R_1 occurs during the ascent of R and when R_3 has reached only half of its height The peak of S_1 occurs when the descent of R is only half completed and the total duration of S_1 is represented in the descending portion of R_3

Einthoven's nomenclature in this record would apply the name

tive to be easily applied and may be useful for general purposes but they make it impossible to refer to the extra peaks specifically or to measure them. It is therefore suggested that when more

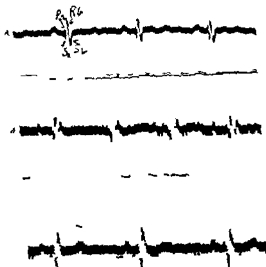


Fig. 90. Record illustrating the suggested terminology for QRS groups which are difficult to measure owing to the present unstandardized nomenclature for the waves.

A. Arbitrary type of QRS with two upward and three downward peaks which may be named as indicated.

B. Shows three upward and three downward peaks constituting a more complicated form of arbitrary QRS group. The downward peaks would be called Q, Sa and Sb, the upward peaks Ra, Rb, Rc.

C. A common form of the M complex. The peaks may be designated as Ra, Rb and S.

than one upward deflection appears the first should be called Ra and subsequent ones Rb, Rc, etc., as occasion arises. A downward deflection preceding the first R would be referred to as Q, and the first downward deflection following the first upward deflection would be called S if there were only one, and Sa if there were more than one. Subsequent downward peaks would be called Sb, Sc, etc., as in Figure 90.

In describing the curves obtained by precordial leads the same arbitrary system of terminology of QRS can be applied with

to take into account the potential contributing to the ascending and descending limbs of the deflections It differed from Einthoven's earlier ideas in avoiding any suggestion of giving the same name to related potentials in different leads It was a purely arbitrary terminology which carried only an implication of direction whether upward or downward and in the case of the terms Q and S of time relative to the upward deflection which was always called R

Although Einthoven's method was more widely used at first the majority preferred to follow Lewis' frankly arbitrary method of naming the individual peaks rather than Einthoven's more superficially logical one Although some have objected that Lewis' method also may interfere with an understanding of the events of the QRS group because the same potential may cause peaks of different names in different leads yet such confusion should not arise if the nomenclature is understood to be entirely arbitrary and not to imply any relationship between peaks of the same name In such an arbitrary system there must not be any implication that similar names have a similar clinical significance for then in occasional cases when the clinical significance is not the same we find ourselves in a quite illogical position Because Einthoven's method as he first used it implied a similar origin for similarly named peaks and because it can be demonstrated that such peaks in different leads of a record do not usually have a similar origin it seems best to avoid Einthoven's method of naming these peaks and follow Lewis' simpler and more arbitrary method of applying Einthoven's letters

Neither of these authors continued to expand his terminology sufficiently to indicate its application to certain unusual varieties of QRS which are encountered It seems necessary that this should be done in order to avoid further confusion and therefore an expansion of Lewis' modification of Einthoven's terminology of the peaks of the electrocardiogram is here submitted

The QRS group A QRS group with two upward peaks separated by a downward one has commonly been referred to as an M shaped complex Likewise when there are two or more upward peaks and two or more downward ones the term vibratory QRS group has often been used These terms are sufficiently descrip

In a group of 17 such records selected from a large clinic series 7 were from patients with disease of the mitral or aortic valve or both and the remainder from patients with coronary arteriosclerosis or hypertension Furthermore Durant who called such a QRS group Q has found that if patients with such records are excluded from a group in which Q_1 is large more patients with valvular disease will be excluded than will remain

In spite of this many physicians have called such waves Q considering that they warrant this title because they are the first downward deflection of QRS Neither Einthoven nor Lewis used the letter Q in this way both of them designating Q as a downward deflection which preceded R It also has been suggested that these waves should be called Q because they often are found in clinical conditions which are likely to be associated with well marked Q waves followed by an R This reason violates the principles of an arbitrary nomenclature In view of the present divergent usage in naming these downward QRS deflections it seems very important to reach an agreement This downward deflection like any other QRS group contains the elements of the Q R and S waves which may appear in other leads of the record It seems advisable in view of the current lack of agreement concerning the name for such deflections and in order to distinguish them from other downward deflections whose apices are followed by an R wave or are preceded by an R wave to call these deflections by the name QS This will indicate that they contain the elements of both peaks and that there is a possibility that in records taken at another time a small R might appear to indicate definitely which name might properly have been attached to the wave Moreover it may be possible by thus separating these records from those with a definite Q (followed by R) or a definite S (preceded by R) to come to understand more clearly what may be their clinical significance

Emphasis must be laid upon the absence of any constant upward deflection no matter how small *preceding this downward peak* If such a deflection is present as in *Figure 12 C* the downward peak must be called S If a small upward deflection follows it as in the first cycle of *Figure 12 A* it must then be called Q In a record such as this *where a majority* of the other downward

equally satisfactory results if it be clearly understood that here the peaks of the same name are even more dissimilar in origin than in the standard leads Nor need records from patients with congenital dextrocardia lead to confusion for Lead 1 is just as truly a reversal of the ordinary Lead 1 if we call the first deflection Q and the second R as if we called them inverted R and S waves respectively To do the latter is not in our opinion good practice as it implies that the name of the peaks has a definite meaning

There has not been a proper agreement as to the naming of the waves of a QRS group like those in Figure 92 C which show first a very small upward deflection for example 1 mm or less followed by a sizable downward peak and another upward peak Some would disregard the small initial upward peak and call the downward one Q and the second upward one R There is a very real objection to this procedure in that it demands a definition as to exactly what size must be reached by the initial deflection before it may be called R It seems more simple and therefore better to call any upward deflection R and to name the other peaks in proper sequence To call such a QRS an M complex is unsatisfactory because it does not allow of measurements of the peaks

Another point of divergent usage is found in the name given to a QRS which is entirely below the isoelectric level with no upward peak which could be called R (Lead 3 of Fig 63 B and C and Fig 12 D and E) Such a downward peak has previously been called S by the author because in limb leads such records are commonly found with right or left axis deviation of QRS and because the downward peak in Lead 1 with right axis deviation (Fig 22 A) and in Lead 3 with left axis deviation (Figs 59 B and 72 B) is usually an S This S is often preceded by a small R as in Figure 12 C and such an R may disappear during one phase of respiration leaving only a downward deflection It is because of the frequency of such records and because they are often associated with ventricular hypertrophy due to valvular disease that the author has previously advocated the use of the letter S for the peak of a QRS group without an upward deflection Such records are almost as commonly obtained from patients with valvular disease as from patients with coronary arteriosclerosis

observed in S of Figure 17 B and in R and R_3 of Figure 17 C Since this is a uniform thickening of the line it may not be comparable to those thickenings which occur in *only* a *portion* of one side of a wave *but until* more definite knowledge on this point is available it seems best to apply the term slurring to this feature As QRS comes to an end at *the* R T or S T junction the slope is often more *gradual* and *the* line more slurred than in other portions of the peak This is seen in R of Figure 36 C ... or at the begin...

so that the situation of slurring must be noted as ... deciding upon its significance It should be described *in a* manner *analogous* to that used for notching namely that it is found a certain number of millimeters from zero and on *the ascent or descent* of a certain wave In the present state of our knowledge it is possible to attach more definite significance *to slurring which does not occur* at one of the three normal sites than to that which does We cannot distinguish the borderline between a *normal* and an abnormal degree of slurring in these normal situations

Measurements of duration The term duration of the QRS group has not been subject to misinterpretation but insufficient attention has been paid to the fact that the duration varies in different leads A *reference to Figure 91* will show that the duration of QRS is shorter in Lead 1 of this record than in Leads 2 and 3 Since this measurement is used to gauge the duration *of the* spreading of the *contraction wave over the* ventricular muscle the longest measurement must be the nearest approach *to* the actual duration of the spreading A shorter measurement which may be obtained *from any one* lead must result from a portion of *the* QRS group being isoelectric in this lead as in Lead 1 of the figure Inasmuch as different records will be found to show the *longest measurement* in different leads it is obvious that no one lead can be selected *to indicate* the duration of the QRS group and therefore all leads should be measured and the longest measurement should be used The duration of the R wave alone will of course have little physiological significance

Similar considerations lead *to the* conclusion that measure

peaks are called S it is best to apply this name also to an occasional deflection such as the one here labeled Q In records which have a small R appearing and disappearing with respiration the disappearance of R may lead to a change of the name of the downward deflection as in Figure 12 A In order to avoid this it is advisable to retain throughout the lead whichever name Q S or QS would belong to the downward deflection in the majority of the complexes It would for example be called S in Figure 12 C because R is found in the majority of the complexes

The use of the term notching should be restricted to doubled or split waves which do not have the peak of the notch so deep as to cross the isoelectric level (Fig 35 A and B) Should the isoelectric level be crossed the term notching should no longer be used the three resulting peaks each taking a name of its own (Fig 92 A and C) A description of the situation of notching in the course of the QRS group is important and should be exactly described It should be stated that it occurs at plus or minus so many millimeters on the descent or the ascent of the wave and the lead should be designated for example at -5 mm on the descent of S This exact description is needed for it has been observed that notching of QRS in certain situations in relation to the peaks is commonly associated with myocardial damage

Lesser grades of disturbance of the course of the ascending or descending limb of a peak are called slurring With this in mind the normal form of the peaks of QRS must be especially noted often the first deflection as it turns away from the P R level does so in a rather gradual slope and the line at this point is broader and therefore more slurred than the subsequent course of the wave which becomes more abrupt and steeper in its further course toward the peak as seen in R_1 of Figure 11 A At the peak of a wave the rate of ascent or descent usually is less abrupt than in the remainder of its course so that one often sees just before or after the peak or perhaps in both situations a thickening of the line to which the term slurring may be applied as in R_1 and S_4 of Figure 17 B In other records a deflection of small size will show a rather uniformly thickened line when compared to the thinness of the line producing larger deflection This may be

observation made by Lewis in 1912 but little noted by most recent authors The deflection of the P R level is caused by auricular activity which continues although diminishing for 0 36 *second* or more after the beginning of P so that this in effect establishes a new zero level for the deflections of the QRS group and the S-T junction from which new level their deflections should be measured Usually this deflection is less than 1 mm but in certain records it may reach a magnitude which makes it important

The T wave Having described the position of the S T junction in relation to this zero we are left with the description of the T wave including under this title the whole of the final deflection of the electrocardiogram It is commonly a simple wave whose first limb is irregularly convex toward its base that is it does not follow the arc of a circle and often shows a more or less straight portion

The term diphasic has been commonly applied to certain peculiar forms of the T wave but there has not been general agreement as to what this term should imply In general a movement is said to be diphasic when it has a positive and a negative phase or vice versa in reference to any given point It would make a difference then whether we consider the deflections of T in relation to the zero level of the record or to the beginning of T itself that is the S-T junction which point might be at zero or above or below it Since electrocardiographic deflections usually are considered as upward or downward in relation to the zero level of the record it seems best to relate the T wave to this level also If there is an apex above the zero level and also one below it the wave may be described as diphasic and the signs + and − may be used to indicate the order of occurrence of the upward (+) and downward (−) peaks that is + − or − + An occasional T wave may be found which shows as many as three apices and such a wave might be called triphasic The term diphasic however will be found applicable to waves of such varied forms that it does not afford a description of T which would be satisfactory in sorting out waves of similar appearance Since a more detailed description is desirable it is recommended that the use of this term be amplified as described below

ments of the duration of any part of the electrocardiogram should be made in all three leads and that the longest measurement should be taken to indicate the actual duration This applies to the P wave the P R interval to QRS and the Q T interval and to the T wave (see Chapter 11)

The S T junction The QRS group passes over more or less abruptly into the T wave The portion of T immediately following the R T or S T junction has often been referred to as the R T or S T interval or segment For purposes of nomenclature we may *speak of these as the S T junction or segment irrespective of what name may be applied to the last peak of QRS* just as we speak of the P R interval irrespective of the name of the first peak of QRS The ending *of the S T segment cannot be definitely indicated unless it be extended to the peak of T for it is actually the first limb of the T wave It seems best from the point of view of nomenclature to avoid the term S T segment and to refer to the whole of the final deflection as the T wave and to speak of its beginning as the S T junction*

In most electrocardiograms the S T junction is effected by a definite change in the inclination of the line of the record toward the isoelectric level so that within less than 0 01 second the R or S wave may be said to end and the T to begin forming a more or less sharp angle at this point Other electrocardiograms may show a gradual *change in the inclination of the line of the record so that R or S may curve gently into the beginning of T sometimes taking 0 04 second or more to complete the transition These two types of S T junction are not separate and distinct but represent the two extremes of the various forms which may be encountered and accordingly various degrees of sharpness of the resulting angle at the junction will be observed One or more leads of the record may show a sharp S T junction and the remainder a less sharp junction If however several cycles are carefully inspected it is usually possible as a result of the variations in the complexes which occur with respiration to discover in even the most gradually curved transition a point which may be called the S T junction*

This junction should be described as being positive or negative in relation to zero Special attention should be given to an

There are records in which it is difficult to mark the ending of T and in which this wave may appear to be prolonged and diphasic or triphasic as in Figure 74 A Lead 3 and Figure 74 C Lead 8 This appearance usually is due to a fusion of T and U and the fact can be recognized if the duration of T is measured in another lead in which T shows a more definite termination If this measurement of duration be applied to the doubtful lead it will be seen that what was thought to be the prolongation or the final apex of T has really occurred after the end of this deflection and is therefore due either to the beginning of U as in Figure 74 A or to the peak of this wave as in Lead 1 F of Figure 77 B

The P wave The P wave may be described in a manner analogous to that proposed for T It may be directed upward or downward or may be diphasic or isoelectric and may show notching of one of its limbs or of the peak Its description and measurement should follow the general lines suggested for T

Clinical Application

For clinical purposes it is unnecessary to work out the basic potential changes as described in this chapter These changes all lead to variations in the direction or form of the waves of the electrocardiogram in the three leads The investigator who wishes to determine the significance of newly recognized abnormalities in the form of the electrocardiogram must have these facts clearly in mind but the analysis having once been done need not be repeated each time the same abnormal form of the record is met with Like variations in the heart s current will always produce like abnormalities in the three leads so that the clinician need only be able to recognize the abnormalities appearing in the form of the curves obtained by the three leads

Notching may result from the sudden addition of the potentials of a relatively large area of muscle newly affected by the contraction wave and it seems likely that this is the origin of the notches at the peak of the curves obtained after a lesion of one bundle branch (Figs 23 B and 24 B) The potential from the first ventricle to contract is suddenly modified by the potential developed when the contraction spreads to the fibers of the other

Having noted the position of the S T junction as + or − or isoelectric we must next indicate the number of apices shown by the T wave using the terms monophasic, diphasic or triphasic We must now describe the character of the curve between the S T junction and the first apex of the T wave It may be concave toward zero horizontal or convex toward zero, or it may occasionally leave zero at an angle in a quite straight line If the line is at first horizontal it always changes to a curve before reaching the apex and the direction of the curve is determined by that of the peak so that this curve need not be especially described The first peak of T should be described as + or − and measured in millimeters If no peak appears, T should be described as isoelectric If more than one apex appears these additional apices should be spoken of as Ta Tb and Tc and their relation to zero indicated as + or − so many millimeters The curve following the first apex of T need not be described for it is so dependent for its form upon the position of the different apices in relation to zero and to the beginning of T that it follows from a description of these points

The description of the T wave will then comprise the following

S T junction (deflection) + 0 − mm
T wave isoelectric monophasic diphasic
triphasic

Course toward first apex of T: horizontal / concave (toward zero) / convex (toward zero) / straight

Apices T or Ta / Tb / Tc (deflection) + 0 − mm

As examples T_3 of Figure 65 A would be described as follows S T junction +8 mm T monophasic course convex +12 mm For T_1 of this figure the description would be S T junction −2 mm T diphasic course convex Ta −3 mm Tb +1 mm For T_3 of Figure 65 c the description would be S T junction +2 mm T diphasic course convex Ta +3 mm Tb −2 mm A more difficult T wave to describe is found in Lead I F of Figure 29 c S T junction +0 2 mm T diphasic course convex Ta −0 7 mm Tb +0 3 mm

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ventricle Large lesions of the Purkinje system or large areas of myocardial degeneration also may produce notching of QRS the electrical effect of a large area of muscle failing to occur at its proper time Notching at or near the peaks of the QRS waves in their large leads usually is due to an abnormality affecting Purkinje tissue or ventricular muscle Notching near the base line in large leads or near the peak in small leads usually is not due to muscle abnormality

An amplification of the nomenclature of the waves of the electrocardiogram has been suggested which affords a detailed analytic description and it is hoped that it may lead to an agreement as to terminology which may facilitate further studies of the significance of the different features of the record

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CHAPTER X

DESCRIPTION AND OPERATION OF THE ELECTROCARDIOGRAPH

THE STRING GALVANOMETER

THE essential units of an electrocardiographic outfit of the string galvanometer type are seen by reference to Figures 93 and 94 to be

1 A source of light (*17*)

2 The galvanometer itself (*N* and *S*)

3 A camera designed to record photographically the movements of the shadow of the galvanometer string (*9* *11* *12* *13*, *15* and *16*)

4 A device for recording units of time along with the string movements so that the duration of the waves may be measured (*18*)

5 A system of resistance coils and switches to facilitate connecting the patient with the instrument and standardizing the galvanometer (*R1* *R2* *R3* *L* and *PR*)

The physical principle upon which the Einthoven *string galvanometer* is based is the same as that used in the electric motor In each instrument movement is produced by the action of one magnetic field upon another There is a very powerful electromagnet produced by coils of wire carrying a current from storage batteries or some other steady source of current The coils are placed about an iron core which ends in large *iron pole shoes* (*N and S Figs* 93 and 94) These point towards each other and are tapered so as to concentrate and intensify the magnetic field

The moving part of the instrument is a very *fine filament* called the string (Fig 93) which carries the *current to be tested* It is *made* of quartz with a thin plating of *silver or gold*

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Wolferth C C and Wood F C The prediction of differences between precordial leads CR CL and CF based on limb lead findings *Am Heart J* 20 12 1940

ing its two ends closer together A slackened string will always move more than a tense one with the same current

The movements of the string in this galvanometer are too small to be recorded without magnification so the pole shoes are bored to allow for the insertion of microscope tubes (Figs 93 and 94 3) One of these contains a condenser objective to bring the light to a focus upon the string and the other an objective for magnification of the shadow of the string and an eye piece for its projection upon the photographic recorder

Many models of the string galvanometer are on the market at present The first made in this country was designed by Professor Horatio B Williams of Columbia University made at first by C F Hindle and at present by the Cambridge Instrument Company of London and New York Figure 94 is a model of the instrument made by Hindle about 1923 The present models are smaller and more simple in construction and a portable model is available which weighs about 30 pounds Satisfactory instruments are also made by other manufacturers

The instrument shown in Figure 94 is a typical apparatus and can be used to explain the principles of operation which are common to all We can see the coils of wire (1) which make the electromagnet the large tapering pole shoes N and S held apart by brass wedges (2) placed in the angles on each side the ends of the microscope tubes (3) the tubular brass device called the string housing (4) which is placed vertically above and below the space between the pole shoes and is used to hold the string in proper position in the magnetic field and to vary its position and tension as is necessary If a battery is used to activate the coils of the magnet an ammeter should be available to make certain that it is properly charged

In order that the microscopes may be properly aligned they are each provided with a three screw centering device (6) similar to that used on some microscope stages The new models find this unnecessary because of a different construction of the string housing

The string housing has two objects in view It must allow the adjustment of each end of the string so that the latter will pass through the center of the magnetic field and must allow the

on its surface The string is about 0 002 mm in diameter and may have an electrical resistance of from 1000 to 15 000 ohms For clinical use the most satisfactory resistance is about 10 000

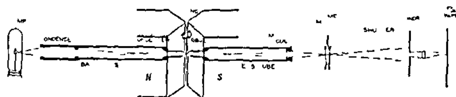


Fig 93 Schematic arrangement of string galvanometer electrocardiograph The blocks N and S indicate the two pole shoes of the magnet It is possible that the polarity might be reversed when the instrument is in use but if it is as represented a current passing through the string from above downward would induce a magnetic field about the string in the direction of the small dotted arrows around the upper portion of the string This would move the string backward away from the observer

ohms Low resistance strings accentuate the tendency to over shooting

The string is held in the center of the magnetic field between the narrowed edges of the pole shoes of the magnet It runs in a direction parallel to these edges as can be seen in Figure 93 where the pole shoes are marked N and S respectively to denote their magnetic polarity A current passing from above downward through the string will induce a magnetic field about it with the direction shown by the dotted circle about the upper part of the string while a current passing from below upward will in duce a magnetic field about the string in the opposite direction In the case of a current from above downward the interference of its induced magnetic field with that between the pole shoes will be on the side toward the person who is looking at the illus tration for the flow of the field between the pole shoes is from *N* to *S* The movement of the string in this case will be away from the observer out of the plane of the page If the current passes in the other direction through the string the movement will be toward the observer because the magnetic field induced about the string is reversed and the interference is on the oppo site side

The movements of this filament will be greater as the current is stronger and as the tension of the string is slackened by bring

string to be tightened or loosened at the will of the operator The first of these objects is attained by the small screws (7) which acting against springs allow us to move the compressed fiber block to which each end of the string is attached in any direction in a horizontal plane This sort of adjustment is provided for both upper and lower ends of the string In the newer models this adjustment of the position of the ends of the string is not necessary because the string falls into its exact position due to the exact machining of its metal case To tighten and loosen the string the wheel (8) is used Turning this acts upon the block with the upper end of the string attached moving it upward or downward as the wheel is turning in a direction against or with the hands of a clock Since the lower end of the string is fixed the string will be tightened when the upper end moves upward and loosened when it moves downward

The string itself is attached at either end to a small spade like piece of brass held in a metal piece which is insulated from the rest of the galvanometer by the above mentioned compressed fiber block and connected to the binding posts on the back of the string housing (4) The housing is mounted on a brass plate upon the back of the pole shoes by means of a three point suspension designed to hold it rigidly in place It is pressed against this brass plate by a large spring

The recording camera has a box (9) to contain a roll of film or bromide paper and another box (10) into which the film or paper passes after being exposed Between these two is a device for making the exposure and for moving the film at a uniform rate The light is admitted through the horizontal slit (11) and an adjustment may be provided for making the slit narrower or wider Behind the slit is a cylindrical lens which focuses the light coming through the slit upon the photographic film behind This cylindrical lens focuses the light to a line which extends across the width of the photographic film and the shadow of the string appears as a break in this line of light Rulings upon the lens throw their shadows on the film as it passes thus making the horizontal lines of the records (Fig 1)

The film passes between two cylinders below the level of the lens which are not in contact except when the lever (12) is moved

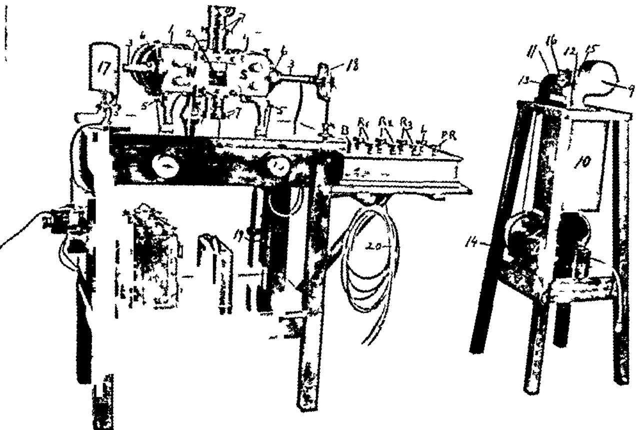


Fig. 91 Early model of the Williams Hindle electrocardiograph

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One of these cylinders is constantly revolving at a uniform rate being turned by the shaft (*13*) which is driven by a small motor (*11*) with a self governing device to keep its speed uniform This motor can be run on the house current The speed is controlled by the operator and set at the desired point by varying a resistance in circuit with the armature of the motor The speed remains as set with great constancy

As the lever (*12*) is moved a jerk is felt when the two cylinders make contact gripping the film between them We can be sure that the film is moving by glancing at the wheel (*15*) attached to the spindle on which the film is wound As the lever is moved farther a click is heard denoting that the shutter behind the slit is opened and that the film is being exposed By moving the lever far enough to start the film and not far enough to open the shutter unexposed film may be run into the lower box

The pulleys on the shaft of the motor and those on the driving shaft of the camera are of different sizes so that the successive speeds at which the film may be propelled past the camera slit are each double the next slower one being about 12 5 25 50 and 100 mm of film per second as the motor is usually regulated The smaller models have only one speed 25 mm per second which is the standard for clinical records

Above the slit (*11*) is a device (*16*) for photographing numbers on the film behind It is an ordinary photographic shutter set for a bulb exposure The numbers are on two celluloid discs which may be revolved at will The numbers are exposed by pressing on the lever that opens the shutter allowing the shadow of the numbers to fall on the end of the film behind

Directly upon the top of the camera table is a sliding knife which must be pulled out before the lever (*12*) can be turned to start the movement of the film The knife is so arranged that it pulls out a slide opening a space in the top of the box (*10*) to allow the film to pass through After the film is exposed the knife should be pushed in thus cutting off the film and allowing it to drop into the lower box The slide in the top of this box must be closed before the box is removed from the camera table

The source of illumination originally used was an arc lamp but since the invention of a more brilliant filament bulb lamp

this has entirely supplanted the arc for clinical purposes There are one or two types of filament lamps available which can be used with perfect satisfaction for clinical work when the speed of the record is not to be over 25 mm per second They will take readable though underexposed records at 50 mm per second With the smaller models records can be taken at 100 mm per second using the filament bulb as a source of light Though the [illegible] mm bulbs must be replaced occasionally because of loss [illegible] proper handling [illegible] amp housing (17) is provided which allows vertical and lateral adjustment of the bulb by screws and has the necessary lenses attached

The time recorder We must have a time marker of some sort recording upon the photographic film both to serve as a check of its speed and to facilitate time measurements in the records The simplest form is a tuning fork placed upon the camera table so that the shadow of a pointer attached to one of its tines will fall vertically across the slit in front of the cylindrical lens This produces a series of up-and-down waves of the period of the tuning fork

A Jacquet chronograph may be similarly placed with the lever vertical and will produce a line upon the record with a small peak every 1/5 second If the lever be placed horizontally between the source of light and the galvanometer and its width is such that the shadow fully covers the slit of the camera it will produce a line of shadow across the film as the lever jumps upward and another as it falls downward Many of the illustrations in this book are records taken when using a timer of this sort The interval from the first one of a pair of lines to the first one of the next pair is 1/5 second The interval between the lines of different pairs is a variable one depending upon how high the lever goes after crossing the slit on its upward fling

Vertical lines across the film are most useful for the purpose of measuring the records and most instruments on the market at present carry a timer throwing a shadow on the slit at 0 01 second intervals every fifth line being accentuated slightly so that the space between the accentuated lines is 1/5 second Many of the illustrations show this sort of a time marking which

when combined with the horizontal ruling and a film speed of 1 mm per 0 01 second gives a system of squares like those of plotting paper This timer is shown in Figure 94 (18) and consists of a rotating wheel having four thin spokes and one slightly thicker The shadow of the thin spokes is just sufficient to cover the width of the slit of the camera As these pass by the slit and cover it the light is cut off for an instant so that the film is not exposed to the light and one of the finer vertical lines is made The thicker spoke makes the fifth and wider line because this shadow lasts for a longer time This time wheel is driven by a small motor governed by a tuning fork of fifty vibrations per second (19) The motor makes one revolution to 10 vibrations of the tuning fork and as the spoked wheel is on the shaft of the motor it is driven at a rate of five revolutions per second

The resistance box When the patient s extremities are connected with the galvanometer through the resistance box there occurs a deflection of the string which is due to the difference in potential existing at the contacts of the patient s skin with the two electrodes This is called the skin current and is sometimes very small amounting to only 1 or 2 millivolts but usually it is between 5 and 10 millivolts and it may be 20 or more If it is large it will cause the string to move so far from the center or zero position that it will be quite off the photographic film and may be outside the circle of light thrown by the microscopes

The objects of the resistance box are (1) To protect the string from being forced too far and perhaps broken by this skin current , (2) to enable the operator to bring the string back to the center of the field of light by passing through it a sufficient current in an opposite direction to neutralize the skin current (3) to allow the operator to produce in the circuit of the patient and galvanometer an exact potential of 1 millivolt in either direction or of multiples of this unit to be used in the process of standardizing the string

The circuit within such a box is shown in diagram in Figure 95 and the same lettering is used in Figures 94 and 96 which show the outside of the box as made by Hindle The same essential features are found in the simplified control panel of the newer models *A* is a dry cell which furnishes the current *B* is

the pole shifter which enables the operator to pass a current in either direction through the rest of the circuit *G* is a small ammeter which indicates the amount of the current The resistance *R1* is varied until the ammeter registers 10 and the current through *R1* then will have a value of 0001 ampere The resistance *R2* forms a derived circuit and the introduction of 10 ohms resistance at *R2* will cause a difference of 001 volt across the terminals of this circuit For each added 10 ohms there will be an added millivolt difference

At *PR* are the resistance coils which form the protecting resistance When the needle of this switch points to *INF*, the circuit between the box and the galvanometer is open and the string is protected from all outside currents With the needle at *2* 1 000 000 ohms are placed in circuit with the patient and galvanometer with the needle at *1* there are 10 000 ohms and at *0* there are none The high resistances protect the galvanometer string as the patient is turned in from the full impact of a large skin current or of any extraneous currents which may be in the circuit through faulty insulation or poor contacts The current will be reduced by the large resistance and the string kept from deflecting outside of the circle of light

The wires from the three extremities of the patient are led into the box at the binding posts marked *RA* (right arm) *LA* (left arm) and *LL* (left leg) The lead switch *L* marked *1 2 3 4*, is a device for connecting these wires to the galvanometer in such a way that when the needle points to *1* Lead 1 is properly connected (*RA LA*) when the needle points to 2 Lead 2 is connected (*RA LL*) and when it points to *3* Lead 3 is connected (*LA LL*)

When the needle of this switch points to *4* the patient is disconnected from the galvanometer and the resistance *R3* is substituted for the patient This resistance is used for measuring by comparison the resistance of the patient it may also be used to measure the resistance of the string * In newer models contact *4*

* To mea ure the resistance of the string the galvanometer is turned on the lea l w tch s turned to contact *4* an l the protecting resistance to zero A w en 1 al of 5 m ll olts is thrown

substitutes for the patient a fixed resistance of 2000 ohms The patient s resistance may be determined by comparing the size of the deflection obtained through the patient and through this resistance

The other binding posts along the back of the resistance box are quite plainly marked the two on the left being for the + and — poles of the dry cell which furnishes the compensating and standardizing current

In this resistance box all the coils and contacts are enclosed in a copper lined box all the handles are of metal and connected to the copper lining and this lining can be earthed from the terminal marked Earth so that effective screening of the enclosed circuits is provided from static charges which might be induced by the movements of the hands of the operator or other causes

Installation Most of the modern instruments are supplied completely set up and ready for use or if not adequate instructions are supplied by the manufacturer so that even the novice will experience no serious difficulty Certain things however may be profitably emphasized

All wires used about the instrument except those which actuate the electromagnet should be covered with lead and it is advisable to have these coverings grounded The grounding wire attached to the back of the resistance box should be of ample size, and the frame of the galvanometer should be grounded One of the ends of the string should be grounded This may be done by connecting together the adjacent terminals on the resistance box These groundings particularly the last two will prevent static charges developing upon the instrument or the string so as to draw the string against one of the pole shoes and perhaps break it They are usually an integral part of the structure of the modern instruments If an alternating current is in use in the building there may be difficulty in screening the galvanometer circuit from the magnetic field about the power wires This field is constantly varying and will induce a current in the gal

on the ruled plate above the slit of the camera Resistance is now turned in at $R3$ until the deflection of the string is reduced to exactly 1 5 cm for 5 millivolts The amount of the resistance necessary will be equal to the resistance of the string and can be read off from the dials of $R3$

galvanometer circuit of the same frequency as the alternations of the current producing a continual buzzing movement of the galvanometer string To prevent this the power wires should be enclosed in an iron pipe which will catch most of the magnetic lines of force and prevent their reaching the galvanometer circuit The thicker the iron the greater the protection Similar induction trouble can arise from the wire supplying the motor of the camera or of the timer should these wires pass near the galvanometer circuit or it can arise from the wire to an arc light with direct current if the arc light should flicker

The proper *centering of the optical system* should be carefully attended to When the source of light and the galvanometer microscopes are in proper alignment screwing the projection microscope (*3*) in and out so that the string shadow goes out of focus first one way and then the other should result in a symmetrical widening of the shadow to either side Likewise the edge of the circle of light should go out of focus concentrically and should continue to be circular If the alignment of the system of lenses is improper the string will broaden out to one side when the microscope is screwed one way and to the other side when screwed the other way The light circle will do likewise and will assume an elliptical form With poor alignment it is impossible to get a sharp photographic image of the string The alignment should be corrected by moving the lamp to one side or the other until the string shadow and the circle of light remain symmetrical as the microscope is focused

If lateral movement of the source of light does not correct the alignment so that focusing gives a symmetrical widening of the string shadow and of the circle of light then the trouble must be in the centering of the microscope tubes To align *these is a nice* procedure and it should not be attempted unless one is quite familiar with the structure of the instrument In the modern instruments this is done at the factory and need not be done again

The photographic image may fail to have sharp edges even though it appears sharp to the observer because of chromatic aberration in the lenses The operator should find by experiment a point somewhere between the eyepiece and the camera

at which he can focus the shadow of the string and obtain a sharp photographic image. The visual image at the distance of the camera will of course then be slightly blurred. The visible light rays which we focus by the eye are mostly from the red end of the spectrum, and these converge from the microscope more sharply than the more actinic blue rays which produce the photographic image.

When connecting the dry cell to the resistance box it is convenient to connect its poles in such a way that turning the pole shifter to the right will enable the operator to move the string shadow to the right upon the camera when resistance is introduced at *R2*. The shadow will then be moved to the left by introducing resistance at *R2* when the pole shifter is turned to the left.

When connecting the resistance box to the galvanometer the wires should be so connected, when film is used, that the normal R wave causes a movement in a direction toward the right hand side of the camera slit as one faces it standing by the galvanometer. If this precaution is not taken prints made from the negative must be printed with the film instead of the emulsion against the paper. Prints made in this way do not, of course, have as good definition as when the emulsion lies next to the paper.

When bromide paper is to be used in the camera the wires to the galvanometer should be connected so that the normal R wave causes a movement in the reverse direction, that is, toward the left side of the camera slit, or the record will be upside down.

Taking the record. The patient must first be connected with the resistance box. A flexible cable (Fig. 94, 20) containing three wires twisted together and covered with a flexible metal sheath should be used. In a hospital this cable may lead to a wall plug which connects with a cable running to various parts of the building and records can thus be taken of patients lying in their beds. The development of portable electrocardiographs which can be pushed about on a wheeled truck have now made such wiring of a hospital unnecessary except for buildings of great size. In the office the wires of the flexible cable are attached at one end to the contacts *RA*, *LA*, and *LL* of the resistance box

and at the other by special electrodes to the right arm left arm and left leg of the patient

The position of the patient should be stated as a part of the

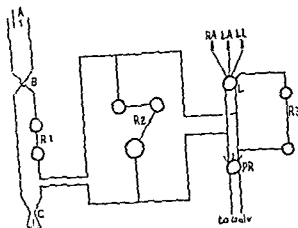


Fig 93 Diagram of the wiring of the resistance box *A* an ordinary dry cell *B* a pole changer *C* an ammeter to measure the current flowing from the dry cell th ou h *R* *R1* esistance on the circuit of the dry cell Wires from the right arm

takes the short circuit afforded by *R2* By introducing resistance here more and more current may be passed through the galvanometer *R3* is an extra resistance which is thrown in circuit with the galvanometer when the lead switch is turned to 4 (Fig 96)

electrocardiographic report because in order to be comparable subsequent records of the same patient should always be taken in the same position as the original If this is not done changes in the electrocardiogram resulting from changes in the patient s position may be erroneously given a diagnostic significance When the record is taken with the patient seated he should not sit slumped down in an upright chair for this will tend to press the diaphragm upward into the thorax and to make the heart lie more transversely The effect of this upon the electrocardiogram has been described When the patient lies flat in bed the effect of abdominal distention or of adiposity will also be to force the diaphragm somewhat abnormally above its natural position in the thorax The effect upon the T wave in certain cases when

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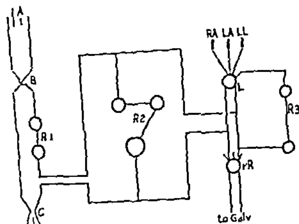


Fig. 95 Diagram of the wiring of the resistance box. *A* an ordinary dry cell. *B* a pole changer. *C* an ammeter to measure the current flowing from the dry cell through *R1*. *R2* resistance on the circuit of the dry cell. Wires from the right arm (*RA*) left arm (*LA*) and left leg (*LL*) come into the lead switch (*L*) by means of which any two extremities may be connected to the galvanometer. The protecting resistance (*PR*) is placed in the circuit to the galvanometer. The current from the dry cell *A* will not flow through the galvanometer with its high resistance, but takes the short circuit afforded by *R2*. By introducing resistance here more and more current may be passed through the galvanometer. *R3* is an extra resistance which is thrown in circuit with the galvanometer when the lead switch is turned to 4 (Fig. 96)

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records are taken in the seated position has been discussed on page 58

To connect the patient with the wires we may use one of several types of electrodes It has been found unnecessary to use the nonpolarizable zinc sulphate cell in salt solution electrode which was at first thought necessary to avoid polarization at the surface of the skin It has been found that a satisfactory contact can be made between the patient and the wires by using tanks containing strong salt solution into which the hands and the foot may be placed and the current led off from the solution by metal plates to which the wires are attached Bandages wet with strong salt solution were formerly applied to the extremities of bed patients, but none of these types of electrode has been found as satisfactory as the electrode composed of a piece of metal about 4 by 6 cm placed upon an area of the skin which has been thoroughly rubbed with a paste containing a large percentage of salt This paste was first devised by Jenks and Graybiel but has been modified and improved by most of the manufacturers since then With proper friction of the skin these electrodes offer a relatively low resistance to the passage of a constant current the resistance measuring by the substitution method is usually less than 2000 ohms Polarization does not seem to affect the usefulness of these electrodes in spite of the fact that technically they are polarizable It is likely that the reason is that the heart's currents are so small in relation to the size of the electrodes surface that what polarization may occur never becomes sufficiently marked to cause distortion of the heart's current

For precordial leads a circular metal contact to which the wire is attached may be placed upon an area of the skin prepared by the electrode paste With sufficient friction and an adequate amount of paste the resistance will be found to be less than 2000 ohms For obtaining esophageal leads an electrode has been described by Hamilton and Nyboer made of German silver 8 mm in diameter by 20 mm in length of circular cross section with slightly tapering ends This is soldered to four strands of insulated copper wire enclosed in a rubber duodenal tube of the Rehfuss type

The patient is thus connected by wires and electrodes with the resistance box at the contacts R 4 I 4 and I L

To take Lead I the switch I is turned so that it points to I

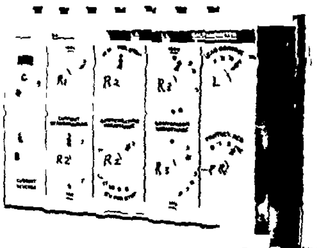


Fig 96 The resistance box The lettering is the same as in Figure 35

the resistance PR is turned from INF to 2 The string shadow should be watched as this is done and it will be seen to jump to one side or the other This movement is due to the skin current At times this current will be strong enough to throw the string shadow completely outside the field of light This is usually due to improper application of the electrodes especially to getting moisture on the contact between two different metals on one of the electrodes or to having one area of contact wetter than the other It may occasionally be a feature of the vasomotor condition of the patient s limbs especially in the presence of marked edema The way to correct it is to reapply the electrodes with care that the skin areas have been well rubbed and have an adequate application of the paste

When the operator observes the direction in which the skin current is deflecting the string he should turn the pole changer B to throw the current into the box in such a direction as to oppose this movement of the string When the dry cell has been

connected as already described, the pole changer should be turned to the left if the string shadow has moved to the right and vice versa. This will not cause any movement of the string as the resistance at *R2* should be at zero. The next step is to turn in sufficient resistance at *R2* to bring the string shadow back to the center of the field of light. With the modern instruments this compensating current is derived from a rheostat resistance which does not produce a measured amount of current but which is turned arbitrarily until the string shadow is forced back to the center of the field. When the shadow is approximately at center *PR* may be turned to *1*, and if the string shadow again moves far off center a sufficient resistance should be turned in or out to bring it again to the center of the camera slit. *PR* is now turned to *0* and the centering process repeated if necessary. This procedure is called *compensating* for the skin current and it will be recognized that what we have done is to turn on sufficient current in a direction opposite to the skin current to neutralize it completely.

The operator is now ready for the next step, namely the *standardization* of the galvanometer so that 1 cm. excursion of the string shadow will represent 1 millivolt in the circuit of patient and galvanometer. The knob of *R2* which gives 1 millivolt units should be turned to and fro so that 1 unit is repeatedly added to and subtracted from the compensating current. With the modern instrument a switch allows either 1 or 3 millivolts to be introduced at once which makes for more exact standardization. The amplitude of the resulting jumps of the string shadow is regulated by turning the wheel (9) on the top of the string housing so as to tighten the string if the jump is too great and slacken it if the jump is too small. When the string has been adjusted so that it moves approximately 1 cm. in response to 1 millivolt then 3 millivolts should be used so that any error in standardization will be multiplied by three and can be corrected by further adjustment of the tension of the string. With the box which has fifteen 1 millivolt steps the string can always be moved 6 cm. directly across the width of the slit, thus making the standardization still more exact.

If the patient is not mentally at ease there may be a continual variation in the skin current and the string shadow will wander back and forth across the field so that standardization becomes difficult The patient should not converse while the record is being taken nor should any other person than the operator be near the patient as the mental reactions of the patient to these things will often cause the skin current to vary We should strive for both mental and physical relaxation

If the plate electrodes are not firmly applied or if the tension on the wires is varied as it sometimes is by *the patient s respiration* movement of the electrode upon the skin may cause an irregular and perhaps quite marked movement of the baseline of the record Similar movement may result from the slipping of the precordial electrode as it is held against the skin of the chest

Contraction of the muscles of the extremities will cause a buzzing vibration of the string which makes it appear out of focus A simple tension of the muscles such as might result from an uncomfortable position will be quite sufficient to cause this The patient should be made comfortable and free from distraction of any kind

Having *compensated* for the skin current and *standardized* so that 1 millivolt causes 1 cm deflection the operator is now ready to *take this lead* Before taking the lead however *it is well* to ascertain the resistance of the patient *With the newer models* this is *measured* by turning the protection switch to *INF*, the lead switch to contact *4* and the compensating current to zero The *protection switch* is now turned to zero and 1 millivolt introduced *in the circuit This* now passes through a resistance of 2000 ohms instead of the patient and if the patient s resistance was 2000 *ohms* the deflection of the string will now be 1 cm, if the patient s resistance was *greater than 2000* ohms the deflection now will be *greater than* 1 cm if the patient s resistance was less than 2000 *ohms the deflection* now will be less than 1 cm *With the older* type of instrument the resistance of the patient is measured by turning *PR* to *INF* turning the lead shift knob to *I* and then *after all the* dials of *R2* are set at *0* turning *PR* to *0* We have now substituted the resistance *R3* instead of

the resistance of the patient and we determine the patient's resistance by comparison with this varying the resistance $R3$ until 3 m[illegible] from $R2$ produces a deflection of exactly 3 cm

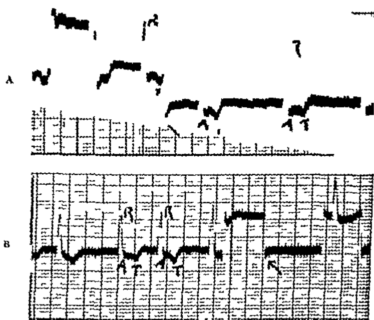


Fig. 97 Two records of Lead I from the same patient to show the deformity of R and T due to overshooting and also that the overshooting diminishes with the skin resistance. The upper record was taken when the resistance was 6000 ohms, the lower when it had fallen to 2000 ohms. The overshooting is seen at the points in the standardization control indicated by the arrows. Note the greater height of R in the upper record and compare the shape of T in the two records at the point indicated by T

The resistance shown at $R3$ then will be the same as was that of the patient

When measured in either of these ways the patient's resistance should not be over 2000 ohms. If it is more than this there will result a *distortion of the electrocardiogram called overshooting* (page 387) which may give rise to a serious error in the interpretation of the record if it is not recognized. The distortion due to overshooting is not as marked with the new small metal plate electrodes and electrode paste as with many of the older forms but may occur even when using the nonpolarizable tank electrode consisting of salt solution in a porous pot standing inside of a zinc sulphate solution from which the current is led off to a

zinc plate That overshooting may be found with such an electrode proves conclusively that it is not due to polarization

The film is exposed by moving the lever (12) on the camera as far as it will go to the right No record the standardization by turning in and then out again 1 or 2 millivolts while the film is being exposed and then allow three or four revolutions of the flywheel which will give 9 or 12 inches of record after the standardization test.

Leads 2 and 3 are taken by exactly the same procedure as employed for Lead 1 except that the lead shift knob is turned to 2 and 3 respectively If it is found that the string tension does not have to be diminished from one lead to the next we know that the resistance of the second of these leads is not more than that of the first. If the first is low it will be unnecessary to measure the resistance of the second for the only important feature of resistance is that it shall not be high In time the operator will be able to observe the overshooting due to high resistance during the process of standardization and then the need for measuring resistance will be past

In order to take the precordial leads it is necessary to disconnect one or two of the wires that have been attached to the extremities and reconnect them If the right arm is used as the indifferent electrode it will only be necessary to disconnect the leg wire and attach this to the precordial electrode The lead switch then may be turned to contact 2 the wires of this lead now having been adapted for the precordial lead In view of the advantages which have been pointed out in favor of the leg as as the site for the indifferent electrode (page 12) it is recommended that having the leg wire attached to the precordial electrode the right arm wire be removed and attached to the leg and then contact 2 will still afford the proper connection for these wires The precordial leads are compensated and standardized by exactly the same procedures as the first three leads the precordial electrode being held against the chest by the patient s right hand which has been freed from the wire It may be repeated here for emphasis that it is advisable to indicate the position of the precordial electrode by the intercostal space and the measured distance from the midline so that if later records

are taken of the patient, the identical point may be used again for the precordial electrode

In taking esophageal leads, if the leg wire is attached to the esophageal electrode and the right arm wire connected to the leg electrode, the records of the ventricular complex will be analogous to those obtained from the precordium in that positivity of the exploring electrode will give an upward deflection and negativity a downward deflection

After the last lead has been taken, and before cutting off the film the lever (*12*) *on the side of the camera should be moved to* the first catch and left so while the flywheel makes two revolutions This runs the end of the record from the level of the slit down to below the level of the knife and leaves a blank unexposed piece of film behind the numbering device upon which to photograph the number of the next record Pressing the knife sharply in will cut off the film and allow it to drop into the box The top of the box should now be closed and the box removed to the dark room for developing

Order of procedure There are so many steps necessary in taking an electrocardiogram that it is well to establish a definite order so that nothing will be forgotten The following will serve as a summary of the taking of a record and will suggest what has been found to be a useful order of procedure

1 Apply the electrodes

2 Turn on the current

3 Photograph the number of the record upon the end of the film or paper

4 Turn the lead switch to Lead 1

5 Compensate and standardize

6 Measure resistance

7 Expose Lead 1 if resistance is satisfactory

8 Turn off protecting resistance and then turn lead switch to Lead 2

9 Compensate and standardize

10 Measure resistance if string has been much slackened

11 Expose Lead 2 if resistance is satisfactory

12 Turn off protecting resistance and then turn lead switch to Lead 3

13 Compensate and standardize

14 Measure resistance if string has been much slackened

15 Expose Lead 3

16 Turn protecting resistance to infinity

17 Attach leg wire to precordial electrode and right arm wire to leg electrode leaving left arm wire in place

18 Turn lead switch to Lead 2

19 Compensate and standardize

20 Measure resistance if necessary

21 Expose precordial lead

22 Turn protecting resistance to infinity

23 Allow paper to run for one revolution of flywheel without opening shutter

24 Cut off paper and close top of box

25 Turn off motor switches of resistance box galvanometer magnet time wheel and light

Overshooting

There can be seen in the standardization test at the beginning of Figure 97 A that there is an overshooting of the deflection due to the millivolt Compare this with the lesser degree of over shooting in Figure 97 B when the resistance was less and with the absence of overshooting shown by the sharp angle at the end of the millivolt deflections in Figure 6 After the overshoot ing the string takes a certain time to return to the level at which it finally remains The time differs in different records some times being very brief sometimes as long as 0 20 second or more Overshooting usually will be more marked the more the string must be slacked to obtain a permanent deflection of 1 cm per millivolt

When the conditions are right for overshooting the standardi zation test and the quick deflections of QRS will show a de flection of more than 1 cm per millivolt and after this deflec tion there will be a slow return toward the baseline distorting the record of the heart s potential during that time Thus the P QRS and T waves will be too large and after each deflec tion the record will tend to overshoot the zero level and curve slowly back toward it Moreover the height of a wave which

begins relatively slowly as does the T wave, will not overshoot so much and so will be relatively less increased than the more quickly developing waves When this artifact is markedly present the end of the T wave will also overshoot the zero and then return to it or if T is downward will curve above it forming an exaggerated U wave

The cause of this overshooting is fairly well understood It does not depend upon the slackness of the string for if the string is left at the same tension as at the time this overshooting was observed when standardizing through the patient and the two ends of the lead wires are short circuited by connecting them together the application of 1 millivolt in the circuit will cause a movement of the string without sign of overshooting This movement will now be much more than 1 cm because of the low resistance of the circuit when the two wires are connected together If the knob for shifting the leads is turned to *1* so that the resistance *R3* is placed in circuit instead of the patient it will be found that a large resistance must be used in this bank to bring the excursion of the string resulting from 1 millivolt to the same size as the deflection when the patient was in the circuit It might be 3000 ohms or more but the application of a millivolt at *R2* through this resistance will not cause overshooting as it does through the patient though the tension of the string has remained unchanged and the resistance of the circuit is now as high as was the resistance of the patient

The cause of the overshooting must lie in the patient I believe that it is the vasomotor condition in the skin under the bandages for it is a rare thing to find overshooting when the patient's extremity is warm and common when it is cold Furthermore I have seen the overshooting disappear suddenly as the cold extremity became warm also accompanied by a change in the skin current When using small lead plate electrodes and the copper wire electrodes described by Stewart I have found overshooting to occur less frequently than when using larger German silver electrodes It is rare to find it when using the modern small plates and electrode paste and is very rare when using tank electrodes It is always found in a patient with cold extremities

because of a weak failing pulse and commonly when edema is present

It is my opinion that the overshooting is due to the vasoconstriction in the skin raising the electrical resistance of this structure so that it functions as a condenser surface The large initial jump is due to the flow of current while the condenser surface of the skin is taking up its charge the more gradual return of the string to its final level is due to the flow of current through from one to the other side of the skin because this tissue does conduct although poorly It acts in this respect like a leaky condenser This hypothesis has not been completely investigated but the many experiments performed are in agreement with it Especially noteworthy is the observation of less overshooting for the same resistance with the smaller electrodes for the condenser surface under the bandage is smaller when the electrode is smaller The skin surface in contact with the water in the usual tank electrode is also small thus accounting for the rarity of overshooting with these

The overshooting can usually be avoided by having the extremities comfortably warm To this end the room must be warm or the patient covered with blankets the electrode paste should be well rubbed into the skin and if the overshooting is still observed a hot water bottle should be placed so as to rest near the part of the extremity to which the electrode is applied Allow from three to eight minutes after applying a hot water bottle for a vasodilatation to take place and the condition will usually disappear Scratching the skin has been suggested but is rarely necessary

Difficulties in Operation

The electrocardiographic outfit needs but little attention to keep it in proper running order Keeping the time wheel and camera motor oiled is about all that has to be done Occasionally however trouble will develop and it is well to know how to locate the most common causes so that the apparatus can be promptly put in order

If on turning in the patient it is found that the string is not deflected as usual by the skin current and the electrocardiogram

begins relatively slowly as does the T wave, will not overshoot so much and so will be relatively less increased than the more quickly developing waves When this artifact is markedly present the end of the T wave will also overshoot the zero and then return to it, or if T is downward will curve above it forming an exaggerated U wave

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object such as a bit of wire or a match across the aperture of the rear microscope (Fig 91 3) Focus the shadow of this sharply Screw the front microscope forward about ¼ inch by means of the focusing adjustment and then screw it back to its former position so that the shadow of the wire is in focus again *If the* string was stuck to the objective of this microscope it will have been dislodged by this process and will reappear *in the circle of* light If not screw the rear microscope backward about ¼ inch and then forward to its former position so that the shadow of the wire is in focus This will dislodge the string if it was stuck to the rear microscope The precaution concerning the shadow of the wire is necessary to prevent each microscope being screwed back past its proper position and against the other one

If the string fails to reappear after the above manipulations of the microscopes it is either stuck to the sides of the slit or is broken Turn the string adjustment wheel for three or four revolutions so as to tighten the string This may pull it free but if it does not now appear in the circle of light it will probably be necessary to call upon the services of a factory trained mechanic or to send the string carrier to the factory for adjustment With *the newer models* of the instrument the string carriage may be easily removed and replaced without danger of breaking the string and this will serve to free it of static charges

The precautions against such sticking of the string are (1) keep the resistance box contacts clean and (2) never leave the galvanometer with the string unusually slackened that is, so that it moves more than 1 cm per millivolt through *2000 ohms* *With* the small model of the Cambridge instrument the string should be left at such a tension that it will deflect 1 cm for 1 *millivolt with no* resistance in circuit

If we find that on turning in the current from Lead 1 *there is no deflection* of the *string* while on turning in either of the other leads there is a deflection *the trouble lies* in the connections with the patient If *Lead 2* is right and *Leads* 1 and 3 are *dead* then the trouble lies in the wire which enters into Leads 1 and 3 and not into *Lead 2* *that is the left arm* wire If the trouble is inside the resistance box there *will be no deflection* of the string on touching moistened *fingers to the binding* posts of the

test another lead to see if that behaves similarly If no deflection is obtained from any of the lead connections turn the switch to contact 4 and attempt to pass current through the resistance coils, as for measuring the patient s resistance If no deflection is obtained through the resistance coils, then the string itself should be tested by touching one moistened finger tip to each of the terminals on the string housing The difference in potential between the two finger tips should cause the string to jump If it does not then the string has ceased to conduct and must be replaced by a new one Obviously if the string does jump when the terminals are touched in this way and has not moved during the previous procedures the trouble must lie in the connections

If the string shows large very rapid irregular vibrations while the dials of the resistance box are being turned it is due to dirt between the contacts of the dials This should be promptly corrected for it may cause the string to be thrown against and stick either to the side of the slit in which it lies or to one of the microscope objectives so that its shadow will not appear in the circle of the light

To clean the contacts of the resistance dials open the box and wipe the surfaces with a fine tissue paper while turning the dials back and forth Do not use emery as the fine grit remains and makes the contact worse rather than better

If the string suddenly disappears from the field of the microscope while the instrument is being used it may have been due to a sudden large variation in the skin current Turn the protecting resistance *PR* to *INF* and the string should reappear If it fails to do so its disappearance is probably due to its being stuck either to the microscope objectives or to the side of the slit This may be due to its having acquired a static charge from defective grounding or to the above described result of dirty contacts in the resistance box To free the string turn off the galvanometer current and turn the protecting resistance to zero which will distribute any static charge still remaining Tapping the microscopes gently with a lead pencil may now dislodge the string if it is stuck to them and its shadow will then reappear in its proper place in the circle of light If this does not suffice a more complicated procedure will be necessary Place some thin opaque

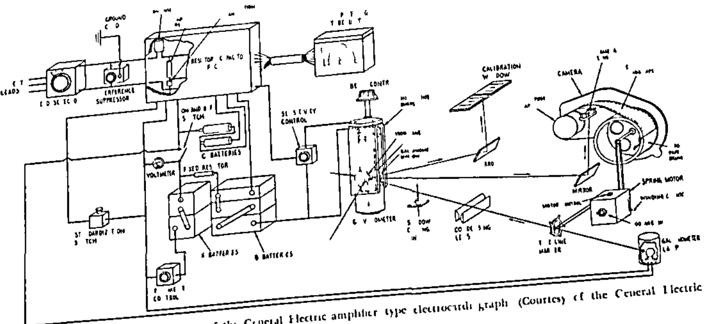


Fig 98 Simplified schematic diagram of the General Electric amplifier type electrocardiograph (Courtesy of the General Electric X-ray Corporation)

wires of the two dead leads the protecting resistance being turned to 0 for example *RA* and *LA* (Lead 1) and *RA* and *LA* (Lead 3) might give no deflection even if Lead 2 is functioning properly If this procedure gives a deflection of the string then the trouble does not lie in the resistance box but in the wires to the patient

Amplifier Tube Electrocardiograph

Of recent years with the development of the amplifier tube it has become possible to record the electrocardiogram by means of an instrument quite different in principle and in method of operation from the string galvanometer Instead of recording the heart s tiny current by means of a very sensitive galvanometer the current from the heart is greatly amplified so that a less fragile instrument with equal sensitivity of its recording part can be used In this new apparatus (Fig 98) the recording element is a beam of light reflected from a moving mirror Several models of this instrument are on the market the first having been developed by the General Electric X Ray Corporation in 1927

One of the main differences between the amplifier and string types of electrocardiograph lies in the principle of recording The string galvonometer is a current recording device whereas the amplifier type electrocardiograph is a voltage recording device In the former the amount of current drawn from the heart is relatively large being determined by the total resistance of the circuit containing the body resistance and the resistance of the galvanometer string In the latter the current drawn from the patient is several hundred times less being limited by a very high resistance The amplifier type electrocardiograph furnishes a direct means for recording heart potentials as contrasted with the indirect means provided by the string galvanometer Therein lies the main reason for the difference in characteristics and method of operation of these two types of instrument

The amplifier type electrocardiograph is independent of body resistance hence no adjustments or precautions in regard to this resistance are necessary This is brought about by the fact that the equipment has inserted in the patient s circuit a resistance of very high ohmic value When the body resistance of the patient which may vary from 1000 to 5000 ohms is added the change is

Fig. 98. Simplified schematic diagram of the General Electric amplifier type electrocardiograph. (Courtesy of the General Electric X-ray Corporation.)

relatively so small that it does not appreciably affect the total. An adjustment of the sensitivity of the instrument in accord with the body resistance is therefore unnecessary.

Compensation for skin potentials is not required and skin voltages even of exceptionally high magnitude cannot damage the equipment or cause a breakage of the galvanometer. Should the recording shadow be deflected by the skin voltage when the patient is connected in the circuit so that it disappears from the calibration scale, it will reappear after a wait of from 10 to 20 seconds and gradually come to rest at the point from which it started. This gradual return of the shadow is due to the presence of condensers in the amplifying circuit which do not maintain the deflection resulting from the impression of a constant voltage such as the skin potential or the millivolt used for standardization.

This instrument is also free from the overshooting due to the capacitative effect of the patient's skin acting like a leaky condenser. The amount of overshooting is dependent on the current taken by the instrument. Since this is several hundred times less than that taken by the string galvanometer such overshooting is proportionately smaller.

Exactness of recording of heart's current. Figure 99 A is a control curve of an early model of the Victor Electrocardiograph and shows the deflection of 1 cm. resulting from the introduction of 1 millivolt difference of potential in the circuit of the galvanometer. It will be seen that as soon as the deflection reaches its peak there begins a slow return toward the baseline. This deflection recedes 0.85 mm. in the first 0.20 second after the initial jump. The return to the baseline takes the form of a curve so that the complete return to zero takes almost 4 seconds. In recent models of amplifier tube instruments an attempt has been made to eliminate the possibility of error inherent in this decay of the initial deflection. Special wiring methods have altered the constants of the apparatus so that the curve of decay following the introduction of a constant current is very nearly straight during the first 0.16 second after the deflection has reached its peak as is seen in Figure 99 B. The decay of the deflection then takes place with

increasing rapidity and in this test returns to zero after 1 86 *seconds*

The tendency of early models of the amplifier tube instrument

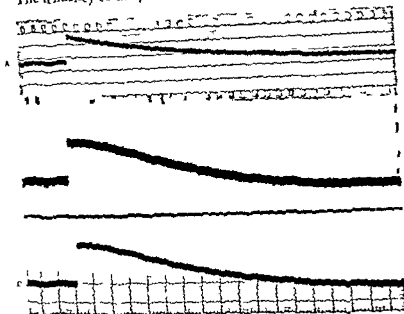


Fig. 99. Records of control curves of amplifier tube electrocardiograph

A Control curve of an early model of the Victor Electrocardiograph showing deflections resulting from the introduction of 1 millivolt potential difference in the circuit of the galvanometer. The deflection very quickly reaches its peak and immediately starts a slow return toward the baseline which it does not reach for almost 4 seconds

B Control curve of an instrument of modern design. Note that after the deflection is completed it remains almost level for about 0 16 second before starting to return to the baseline. The decay then takes place more rapidly being completed in 1 8 seconds

C A similar control curve with the Sandborn instrument. In this curve the decay is not completed until 2 5 seconds after the initial deflection. As in record B the start of the decay is delayed for 0 16 second so that the curve is practically straight during this time

to show a return of the deflection to zero in response to a constantly maintained potential was a source of some apprehension to the users of this type of apparatus for fear that it might result in a distortion of such portions of the electrocardiogram as maintained a level or approximately level deflection. Such electrocardiograms are extremely rare but one may be seen in Figure 100 which shows about the maximum degree of distortion that could

relatively so small that it does not appreciably affect the total An adjustment of the sensitivity of the instrument in accord with the body resistance is therefore unnecessary

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Exactness of recording of heart s current Figure 99 A is a control curve of an early model of the Victor Electrocardiograph and shows *the deflection of 1 cm resulting from the introduction of* 1 millivolt difference of potential in the circuit of the galvanometer *It will be seen that as soon as the deflection reaches its peak* there begins a slow return toward the baseline This deflection *recedes 0 85 mm in the first 0 20 second after the initial jump* The return to the baseline takes the form of a curve so that the *complete return to zero takes almost 1 seconds In recent* models of amplifier tube instruments an attempt has been made to eliminate *the possibility of error inherent in this decay of the initial* deflection Special wiring methods have altered the constants of the apparatus so *that the curve of decay following the introduc*tion of a constant current is very nearly straight during the first 0 16 second after *the deflection has reached its peak* as is seen in Figure 99 B The decay of the deflection then takes place with

low and high frequencies without significant distortion The vacuum tubes are standard radio tubes but especially selected and matched in sets They have a reasonably long life and are easily replaceable

The power for operating the amplifier is provided by dry batteries located in the power compartment of the unit The instrument can therefore be operated anywhere independent of commercial electrical supply The two A batteries supply the power for lighting the filaments of the vacuum tubes and the galvanometer lamp The three B batteries provide voltages for the plate circuit of the tubes The grid bias is derived from the two C battery cells Battery life is reasonably long and their replacement is simple and inexpensive A battery test permits connecting each group of batteries to the voltmeter so that their voltage may be checked at any time

A filament control regulates the voltage impressed across the filament of the vacuum tube and in conjunction with a calibrated voltmeter permits maintaining the voltage at a certain constant value This voltage is also impressed across the standardization circuit when the standardization switch is actuated The circuit consists of two resistances of such value and so arranged that one millivolt can be obtained and impressed on the input side of the amplifier for calibration purposes To obtain exactly one millivolt it is necessary to adjust the filament control so that the voltmeter needle is exactly on the calibration line It is best to test the standardization with the lead selector on position S on which the patient is short circuited If it is desired to record standardization on patient s leads all that is necessary is to operate the standardization switch in the course of recording each lead The magnitude of the standardization deflection remains constant for several tracings or as long as the voltmeter needle rests on the calibration line If the needle drops below this line the filament control should be adjusted to bring it to the original position

The galvanometer consists of two coils of very fine wire wound on small air core spools a phosphor bronze ribbon iron vane and a concave mirror The ribbon is suspended at a definite tension between the two coils which are facing each other and are

be encountered Figure 100 A is Lead 3 taken with a string galvanometer The *S T* segment of this record is more nearly straight and horizontal than any other which has been observed

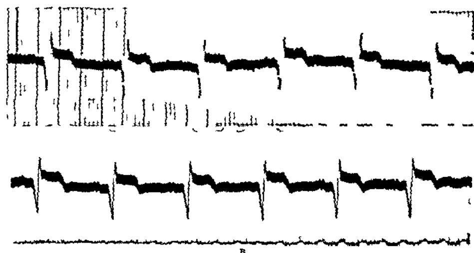


Fig. 100 Two records by Lead 3 of the same patient to illustrate the deformity of the electrocardiogram resulting from the decay of a maintained potential

A Record made with a string galvanometer

B Record made with an early model of the Victor Electrocardiograph A very slight difference in the first phase of the T wave may be observed that in record B being somewhat more inclined toward the baseline than in record A where there is a little more distinct shelf at the beginning of the deflection which returns the S T segment to the baseline

Figure 100 B shows a record of the same patient made at the same time without removing the electrodes but using an old model of the Victor Electrocardiograph made before the circuit was modified so as to retard the first portion of the curve of decay It will be seen that the S T segment inclines downward from the junction slightly more than does the corresponding portion of record 100 A Leads 1 and 2 of this patient showed no differences in the form of the curves obtained with the two instruments No distortion of the usual T wave would occur because there is no section of it which maintains a constant deflection The newer models as has been pointed out have corrected even this source of possible error

In the present model of the General Electric Company's instrument the amplifier is essentially a three stage resistance coupled type especially designed to meet the needs of electrocardiographic work and capable of recording heart potentials of both

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The galvanometer consists of two coils of very fine wire wound on small air-core spools a phosphor bronze ribbon iron vane and a concave mirror The ribbon is suspended at a definite tension between the two coils which are facing each other and are

slightly separated A small iron vane is fastened on one side of the ribbon and the concave mirror on the other The mirror is facing the window through which the beam of light enters the galvanometer The coils and ribbon are mounted in one assembly and immersed in special damping oil Each galvanometer is tested individually for deflection time magnitude of response damping and other characteristics The entire galvanometer is hermetically sealed, remaining fixed in its adjustment indefinitely and not requiring any attention

The heart potentials after being amplified pass through the galvanometer coils, setting up a magnetic field which in conjunction with the fixed field provided by a permanent magnet mounted outside of the galvanometer acts upon the iron vane and causes it to turn one way or the other depending upon the flow of current through the coils The amount of twisting or turning of the iron vane even for the tallest heart deflections is very small not exceeding 10 degrees each way

The optical system is designed to provide a conventional white tracing on a dark background The light source is a concentrated filament lamp mounted in a case The power for lighting the lamp is derived from the A batteries The light is allowed to pass out through a small circular hole in the case It is then reflected by a mirror passed through a pair of condensing lenses before falling upon the galvanometer mirror A steel pin is mounted between the condensing lenses and the galvanometer and its shadow traces the heart record on the paper and also appears in the calibration window The light reflected from the galvanometer mirror falls on a mirror immediately below the camera opening passes through a cylindrical lens mounted in the camera and reaches the sensitive paper A portion of the reflected light is intercepted by another mirror which reflects it upward to the calibration window incorporating a ground glass scale graduated in 5 mm steps This window permits the operator to observe at any time the movements of the shadow from the galvanometer

No adjustment of the optical system is necessary excepting that of the galvanometer lamp when it is replaced Due to the fact that the filaments in these lamps do not all occupy exactly the

same position it is necessary to adjust the lamp by rotating it and moving it up and down in order to obtain an illuminated field as bright and as evenly distributed as possible on the camera mirror and on the calibration window

A beam adjustment is provided to enable the operator to have control of the position of the galvanometer mirror This adju t

mounted on the

by gearing that

ne beam control

knob The rotation of the magnet acts upon the iron vane in the galvanometer and causes it to move in unison thus permitting centering the shadow on the calibration scale

Difficulty which might arise from the pick up by the patient of alternating current fields is solved by the interference suppressor The suppressor is so connected in the circuit that the interference induced in the patient is counteracted by that picked up by the ground cord of the suppressor

A spring motor drives the photographic paper in the camera as well as the time marking device It is a precision motor and is so constructed that it will maintain a constant speed (± 1 per cent) on being wound for a 25 foot run of paper The motor speed can be checked at any time the operator may care to do so by means of a stroboscopic disc and a neon glow bulb provided or by simply determining the time it takes a marked scale to make a certain number of revolutions

The camera is arranged so as to be removable from the unit and has a capacity of 50 feet of paper at one loading The paper is coated with bromide emulsion and has a width of 45 mm The exposed paper can be removed at any time without disturbing the unexposed portion A footage indicator on the panel of the apparatus case informs the operator of the amount of paper remaining in the camera and aids in obtaining tracings of desired lengths

The procedure of operation The following brief description will bring out the method of operating the equipment

1 Attach the electrodes to the patient as usual except that special precautions to insure low skin resistance are not neces

sary. The patient's cord leads are properly labeled and color coded.

2. Place the lead selector switch on position S. This automatically short circuits the patient from the apparatus and makes the instrument ready for calibration.

3. *Remove the main switch plug from "off" receptacle and in*sert it into the "on" receptacle. This turns on the entire apparatus.

4. Allow about 10 seconds for the equipment to stabilize. This is necessary to give time for the condensers of the amplifier to charge and for the vacuum tubes to reach a steady state. When the apparatus is stable, the shadow should have appeared on the calibration window and have become stationary.

5. Press down the standardization switch and note the deflection of the shadow on the calibration window. Adjust the sensitivity control until the deflection is 1 cm. Once set, it will remain constant as long as the voltmeter needle is on the calibration line. If the voltage drops, adjust the position of the filament control switch to bring the voltmeter needle on the line.

6. Start the spring motor by turning the motor knob. This causes the paper to move at the speed of 25 mm. per second.

7. Momentarily press the standardization switch. This makes a record of the standardization on the paper. If the standardization record is desired on the leads, press the switch in the course of recording of each lead.

8. Stop the spring motor.

9. Turn the lead selector to Lead I. Wait for the shadow to come back into view on the calibration scale, which may take from 5 to 20 seconds. The heart action may now be observed. Center the shadow excursions with respect to the center line in the calibration scale by means of the beam control.

10. Start the spring motor and allow as much paper as desired to be exposed. The amount of record exposed will be shown by the footage indicator.

11. Stop the spring motor. Turn the lead selector to Lead 2. Duplicate action as with Lead 1.

12. The other leads are taken in an analogous manner.

13. Turn off the power from the apparatus.

14 Remove the camera to the darkroom for development of the record

Sources of difficulty The operation of amplifier tube instruments is remarkably simple and free from technical difficulties owing to the fact that there is no trouble from the patient's resistance and that compensation and standardization need not be performed with each lead In the majority of cases however the acts of compensation and standardization of the string galvanometer do not take longer than is consumed in waiting for the shadow of the amplifier tube instrument to come to rest at zero Careless operation of the instrument does not however lead to any happening comparable to the breaking of a string in the Einthoven electrocardiograph

There is one feature in some models which will at times be a handicap in the clinical use of the instrument This is the relative narrowness of the recording film or paper If its width is less than 60 mm one will occasionally meet with records giving a *total* amplitude of deflection which is so large that unless the zero level remains quite constant the deflections will shoot off the recording film This difficulty is best met by reducing the sensitivity of the galvanometer to 5 mm per millivolt This procedure will change the appearance of the electrocardiogram though of course not the proportionate size of the various excursions

The chief source of trouble with amplifier tube instruments arises from the appearance of vibrations of the beam of light which occur independently of the heart and are usually due to bad contacts somewhere in the circuit The most frequent situation of this is in the connections of the B batteries and at times in the internal structure of the battery itself Occasionally the contacts of the batteries will become corroded The B batteries should be tested weekly in order to forestall the unexpected appearance of weakness of these batteries When they have run down to a certain extent they may in the course of a few days become wholly unsatisfactory so that the galvanometer instead of remaining stationary at zero will show a continual series of large waves making it impossible to obtain a record

Another feature of the present amplifier tube instruments

sary The patient's cord leads are properly labeled and color coded

2 Place the lead selector switch on position S This automatically short circuits the patient from the apparatus and makes the instrument ready for calibration

3 Remove the main switch plug from off receptacle and insert it into the on receptacle This turns on the entire apparatus

4 Allow about 10 seconds for the equipment to stabilize This is necessary to give time for the condensers of the amplifier to charge and for the vacuum tubes to reach a steady state When the apparatus is stable the shadow should have appeared on the calibration window and have become stationary

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6 Start the spring motor by turning the motor knob This causes the paper to move at the speed of 25 mm per second

7 Momentarily press the standardization switch This makes a record of the standardization on the paper If the standardization record is desired on the leads press the switch in the course of recording of each lead

8 Stop the spring motor

9 Turn the lead selector to Lead 1 Wait for the shadow to come back into view on the calibration scale which may take from 5 to 20 seconds The heart action may now be observed Center the shadow excursions with respect to the center line in the calibration scale by means of the beam control

10 Start the spring motor and allow as much paper as desired to be exposed The amount of record exposed will be shown by the footage indicator

11 Stop the spring motor Turn the lead selector to Lead 2 Duplicate action as with Lead 1

12 The other leads are taken in an analogous manner

13 Turn off the power from the apparatus

one should be able to select the instrument best adapted to the purposes in view

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which might occasionally lead to error is in the method of producing the vertical time lines Because the rotator which causes these lines is run from the same spring motor as is the photographic paper it follows that if the paper slows slightly the time lines will still continue to occur at the same distance apart on the paper although these distances now indicate longer intervals of time The reverse of this will occur if the paper speeds up It is true that such variations in the speed of the paper rarely occur in the course of the taking of a record but experience has shown that it does happen occasionally If the change in speed of the photographic paper is sufficient to cause an observable change in the intensity of the exposure then there will be seen a darker or a lighter portion of the record depending upon whether the paper is running slower or faster than the standard If the paper should run uniformly slow or uniformly fast throughout the record this would not be suspected All such instruments have provisions for checking the speed of the paper and also the speed of the time wheel but unless this checking is done frequently one might miss an occasional period of defective operation of the instrument

In most string galvanometers the timing is effected by a wheel which is electrically controlled independently of the movement of the photographic paper so that should the paper vary in speed the shadows of the spokes of the time wheel will still represent the same interval of time

In regard to the sensitivity or the ability to make an exact record of the electrocardiogram there is at present no difference between the string and the tube galvanometer This matter need not be considered in making a choice Factors which must be taken into account for hospital work are the question of rugged construction the ease of marking records for identification the mobility of the apparatus and the ability to take a number of records without removing the camera box For office and consultation work on the other hand portability is an important feature as is also a rugged construction which will withstand the jars of carrying about One should also take into account whether or not there will be a need to take records where there is not an available alternating current With these qualifications in mind

one should be able to select the instrument best adapted to the purposes in view

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APPENDIX I

THE DETERMINATION OF THE DIRECTION AND THE MANIFEST VALUE OF THE VECTORS OF THE ELECTROCARDIOGRAM

Method of Einthoven
Method of Carter Richter and Greene
Method of Dieuaide

APPENDIX I

THE DETERMINATION OF THE DIRECTION AND THE MANIFEST VALUE OF THE VECTORS OF THE ELECTROCARDIOGRAM

EINTHOVEN'S tables for the determination of the direction within the heart of any current recorded by the three standard leads These directions refer only to the frontal plane so that currents which lie in other planes are only represented as they affect the frontal plane

One must first make certain that the deflections measured in the three leads represent synchronous points of the electrocardiogram for otherwise the deflections will not be due to the same current within the heart This can often be approximated by an inspection of the record measuring with a magnifying glass the distance from the beginning or ending of some sharp deflection such as Q R or S If we are measuring deflections which represent the same current within the heart they will be found to fulfil very closely the formula

Value Lead 1 + value Lead 3 = value Lead 2

If they do not approximate this formula they do not arise from the same current within the heart

The direction of any current within the heart is expressed in relation to the horizontal the 180° below the horizontal being considered positive (+) and the 180 above the horizontal negative (−) The direction 0 is horizontally to the patient's left and ± 180 horizontally to the patient's right (Fig 21) Table XIII is the most satisfactory for ordinary use but Table XII shows rather graphically the reciprocal variation of the lead values with changing direction of the vectors

To use Table XII the recorded potential is determined by measuring the height of the wave at the same time instant in

TABLE XII

SUPPLEMENTARY TABLE

	A Degrees to be Added to the Angle When Column B Is Read Downwards	B Value of the Smallest Lead When the Largest Is Reduced to ±10	C Degrees to be Added to the Angle When Column B Is Read Upwards	D Manifest Potential
Section 1	2	±0 4	8	11 3
	4	±0 8	6	11 1
	6	±1 2	4	11
	8	±1 5	2	10 8
	0	±1 8	0	10 7
Section 2	2	±2 2	8	10 5
	4	±2 5	6	10 4
	6	±2 9	4	10 3
	8	±3 2	2	10 2
	0	±3 5	0	10 2
Section 3	2	±3 8	8	10 1
	4	±4 1	6	10 1
	6	±4 4	4	10
	8	±4 7	2	10
	0	±5	0	10

each lead The value of the smallest lead is then made proportional to a value of 10 for the largest lead by substituting the measured values in the formula

Value largest lead 10 value smallest lead x

Thus if Lead 1 = + 3 2 Lead 2 = + 12 5 Lead 3 = + 9 3 then the formula would be

$$12\,5 \quad 10 \quad 3\,2 \quad x \left(\frac{3\,2 \times 10}{12\,5} = x\right) \text{ therefore } x = 2\,56$$

We now refer to Table XII for the nearest approximation to the values Lead 1 = 2 56 Lead 2 = 10 and we find that this relation of the values lies between 70 and 80°—one might say it was approximately 75

If it is desired to locate the angle with greater exactness than

Table XII

Angle α	Registered Potentials Reduced to a Value of ± 10 for the Largest Wave			Manifest Potential Fм
	Lead 1	Lead 2	Lead 3	
−80	1 8	−8 2	−10	10 7
−70	3 5	−6 5	−10	10 2
−60°	5 0	−5 0	−10	10 0
−50	6 5	−3 5	−10	10 2
−40°	8 2	−1 8	−10	10 7
−30	10 0	0 0	−10	11 5
−20°	10 0	1 8	−8 2	10 7
−10°	10 0	3 5	−6 5	10 2
0°	10 0	5 0	−5 0	10 0
10°	10 0	6 5	−3 5	10 2
20°	10 0	8 2	−1 8	10 7
30	10 0	10 0	0	11 5
40	8 2	10 0	1 8	10 7
50	6 5	10 0	3 5	10 2
60°	5 0	10 0	5 0	10 0
70	3 5	10 0	6 5	10 2
80	1 8	10 0	8 2	10 7
90°	0	10 0	10 0	11 5
100	− 1 8	8 2	10 0	10 7
110°	− 3 5	6 5	10 0	10 2
120	− 5 0	5 0	10 0	10 0
130	− 6 5	3 5	10 0	10 2
140°	− 8 2	1 8	10 0	10 7
150	−10 0	0	10 0	11 5
160°	−10 0	− 1 8	8 2	10 7
170	−10 0	3 5	6 5	10 2
±180	−10 0	− 5 0	5 0	10 0
−170°	−10 0	− 6 5	3 5	10 2
−160	−10 0	− 8 2	1 8	10 7
−150	−10 0	−10 0	0	11 5
−140	− 8 2	−10 0	− 1 8	10 7
−130	6 5	−10 0	− 3 5	10 2
−120	− 5 0	−10 0	− 5 0	10 0
−110	− 3 5	−10 0	− 6 5	10 2
−100	− 1 8	10 0	− 8 2	10 7
− 90	0	10 0	−10 0	11 5

TABLE XIII

THE ANGLE ALPHA

Values of $\epsilon/\epsilon\#$	Largest Lead / Smallest Lead	$e_1=+10$ 2	$e_1=+10$ 3	$e_1=-10$ 2	$e_1=-10$ 3	$e_2=+10$ 1	$e_2=+10$ 3	$e_2=-10$ 1	$e_2=-10$ 3	$e_3=+10$ 1	$e_3=+10$ 2	$e_3=-10$ 1	$e_3=-10$ 2
	E10												
0 00	1 15	−30	30	150	−150	90	30	− 90	−150	90	150	−90°	−30°
0 20	1 14	−29	9	151	−151	89	31	− 91	−149	91	149°	−89	−31
0 40	1 13	−28	28	152	−152	88	32	− 92	−148	92	148	−88	−32
0 59	1 12	−27	27	153	−153°	87	33	− 93	−147	93	147	−87	−33
0 78	1 11	26	26	154	−154	86	34	− 94	−146	94	146°	−86	−34
0 96	1 10	25	25	155	−155	85	35°	− 95	−145	95	145°	−85	−35
1 14	1 09	−24	24	156	−156	84	36	− 96	−144	96	144°	−84	−36
1 32	1 09	−23	23	157	−157	83	37	− 97	−143	97	143°	−83	−37
1 50	1 08	−22	22°	158	−158	82	38	− 98°	−142	98	142	−82	−38
1 68	1 07	21°	21	159°	159	81°	39	− 99	−141	99	141°	−81	−39
1 85	1 06	−20	20	160	−160	80	40°	−100	−140	100	140	−80	−40
2 02	1 06	−19	19	161	−161	79	41	−101	−139	101	139	−79	−41
2 19	1 05	−18	18	162	−162°	78°	42	−102	−138	102	138	−78	−42
2 35	1 05	17	17°	163	−163	77	43	−103	137	103	137	−77	−43
2 52	1 04	−16	16	164	−164	76	44	−104	−136	104	136	−76	−44
2 68	1 04	15	15°	165	−165	75	45	−105	−135	105	135°	−75	−45
2 84	1 03	−14	14	166	−166	74	46°	−106	−134	106	134	−74	−46
3 00	1 03	13	13	167	−16 °	73	47	−107	−133	107	133	−73	−47
3 16	1 02	−12	12	168	−168	72	48	−108	−132	108	132	−72	−48
3 32	1 02	11	11	169	−169	71	49	−109	−131	109	131	−71	−49
3 47	1 02	10	10	170	−170	70	50	−110	−130	110	130	−70	−50
3 63	1 01	− 9	9	171	−171	69	51	−111	−129	111	129	−69	51
3 78	1 01	8	8	172	−172	68	52	−112	−128	112	128	−68	−52
3 94	1 01	− 7		173	−173	67	53	−113	−127	113	127°	−67	−53
4 09	1 01	− 6	6	174	−174	66	54	114	−126	114	126	−66	−54
4 24	1 00	5	5	175	−175	65	55	115	−125	115	125	−65	−55
4 39	1 00	− 4	4	176	−176	64	56	−116	−124	116	124	−64	−56
4 55	1 00	− 3	3	177	177	63	57	−117	−123	117	123	−63	−57
4 70	1 00	2	2	178	−178	62	58	−118	−122°	118	122	−62	−58
4 85	1 00	1	1	179	−179	61	59	−119	−121°	119	121	−61	−59
5 00	1 00	0	0	180	−180	60	60	−120	−120	120	120	−60	−60°

thus the value of the smallest deflection 2.56 mm in this case *must* be located in column B of the Supplementary Table. Each section of this table will be found appropriate to fill in the gap *between certain* figures of the main table though sometimes it must be *inverted* that is read from below upward in order to do so. The Supplementary Table indicates the angle to be added to the smaller value obtained from the main table either in column A or column C depending upon whether or not the figures in the Supplementary Table must be read downward or upward to have them take their place between the figures of the main table. If read from above downward the angle is to be found in column A; if from below upward the angle is to be found in column C.

In column B of the Supplementary Table the nearest value to +2.56 is +2.5 which is found in Section 2. This section must be inverted (read upwards) in order to fit into its appropriate place in Table XII so that the supplementary angle is to be found in column C and is 6°. This angle is to be added to the basic 70° obtained from the main table making 76° which is the exact angle to produce a vector with the designated recorded values (e 1 = 3.2, e 2 = 12.5, e 3 = 9.3).

Should we wish to determine the manifest potential (Em) that is the hypothetical potential which would give rise to such lead excursions we use the figure for manifest potential found in column D of the Supplementary Table opposite the angle obtained from this table. In this case the figure in Section 2 opposite 6° is 10.4. This is the manifest potential for a maximum excursion of 10 mm so that for the lead values with which we started this figure must be changed according to the formula

$$10.4 : 10 :: x : 12.5 \left(\frac{10.4 \times 12.5}{10} = x\right) \text{ therefore } x = 13$$

This is the potential (Em) which would give the lead excursions we have measured if its direction were 76° and there were no short circuiting within the body.

It is well to emphasize again here that this manifest value does not actually exist. Within the heart there are at any one instant many potentials in many different directions. The sum of all

Again if the values are e 1 = 8 e 2 = −4 e 3 = −12

$$\left(\frac{4 \times 10}{12}\right) = 3.33$$

the horizontal line will be opposite 3 32 and the vertical column will be under e 3 = −10 (the lead of the largest value) in the column headed by 2 (the lead of smallest value) The angle is −49° in this case

[illegible] much quicker to use than is Table XII for it does

[illegible] ble XIII is also very simple for the figure in the column under E10 the second column which lies on the same line as the angle obtained is to be used just as was the similar figure obtained from the supplementary table of *Table* XII The largest recorded value in the leads is placed in the formula F10 10 X largest value For the first example above the manifest potential would be 16 16 and for the second example it would be 12 24

Carter Richter and Greene have devised a chart which is reproduced in Figure 101 for the graphic determination of the angle α using the values actually measured in the three leads In order to use this chart to determine the angle the values for each lead are measured or one may be approximated as has been suggested so that they fulfill Einthoven s formula The values measured in two of the leads are counted off from the center of that lead (0) in either the positive or negative direction depending upon whether it is an upward or downward deflection The lines perpendicular to the side of the triangle from the points measured off on the leads in question are followed in to their intersection and a dot made at this point A line the radius drawn from the center of the circle through this point should be projected to the circumference of the circle surrounding the triangle and the angle α will be read off upon this in degrees with the corresponding negative or positive value indicated As an example if the value of Lead 1 = +9 Lead 2 = +16 Lead 3 = +7 the intersection of +9 measured upon Lead 1 and +7 upon Lead 3 is at the point indicated by a dot the line projected from the center through this dot can be seen to strike the periphery of the circle at +55 5° which is the angle α for such deflections Again

these is a certain potential having a certain direction, and this potential, reduced by short circuiting within the body gives rise to the excursions in the leads The manifest potential has a value which is due to the short circuiting of the summated heart potentials at a given instant If there were no short circuiting the current within the heart would be the same as the manifest potential

To use Table XIII to determine the angle of any potential difference within the heart from its registered potential in the leads (e 1, e 2 and e 3) the smallest of these must be made proportional to a value of 10 for the largest (Multiply the smallest value by 10 and divide by the largest value) Compare the figure thus obtained with those in the first column of the table and the angle will be on the *horizontal* line whose number is closest to the figure The angle will be found at the intersection of this line with the *vertical* column determined by the combination of three factors which lead has the largest excursion whether this has a + or a − value and which lead has the smallest excursion For example if the largest deflection is a + value in Lead 1 the vertical column would be under e 1 = +10 either in the column headed by 2 if the smallest deflection were found in Lead 2 or in the column headed by 3 if the smallest deflection were found in Lead 3 If the largest deflection were a − value in Lead 2 the vertical column would be under e 2 = −10 either in the column headed by 1 if the smallest deflection were found in Lead 1 or in the column headed by 3 if the smallest deflection were in Lead 3

For example if the values in the leads were

$$e\,1 = 16 \quad e\,2 = 10 \quad e\,3 = -6$$

then by applying the above formula to the *smallest* and the largest values we obtain

$$\left(\frac{6 \times 10}{16}\right) = 3\,75$$

The angle is therefore found on the horizontal line opposite 3 78 which figure is the nearest to 3 75 Since the largest value was a + value in Lead 1 the vertical column will be under e 1 = + 10 and since the smallest value was in Lead 3 the particular column will be under 3 The angle in this case is 8°

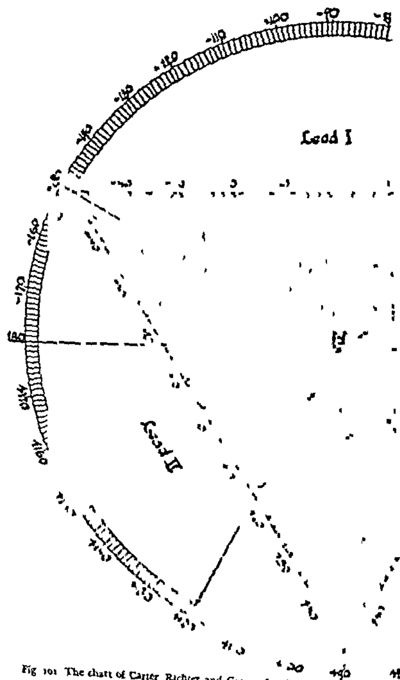


Fig 101 The chart of Carter Richter and Greene for determining the directic

if the values are Lead 1 = −5 Lead 2 = +5 Lead 3 = +10 then at the junction of −5 from Lead 1 and +10 from Lead 3 a dot has been made and the radius of the circle passing through this dot reaches the periphery at +120° which is the angle α for these values Again if the values are Lead 1 = +15 Lead 2 = −1 Lead 3 = −16 2 the intersection of a projection from +15 in Lead 1 and −16 2 in Lead 3 is shown by a dot and the radius of the circle passing through this dot reaches the periphery at −33° which is the angle α for these values

The *manifest value* Em may be determined from this chart also if the distance of the point of intersection of the lead values from the center of the circle be measured off along any line parallel to a lead The distance from the center of the circle to the dot which gave the angle 55 5° if measured along the horizontal parallel to Lead 1 gives Em = +16 2 The distance from the center of the circle to the dot which gives the angle of 120° if measured along the horizontal parallel to Lead 1 gives Em = +10 and distance from the center to the dot which gave the angle 33° if measured along the same horizontal gives Em = +18 Owing to the relatively small difference between the value recorded in the lead and the manifest value it is not as exact to determine this from the chart as it would be from Table XIII

Dieuaide's chart A chart has been devised by Dr F R Dieuaide (Fig 102) which also allows the determination of the angle α by using the actual values recorded in the leads It is exact to within 1 or 2 degrees which is well within the limits of error of the measurements of the waves in the leads In using this chart the measured values may be applied directly as follows *The line of zero for Lead 1* is the horizontal line in the center of the chart marked e 1 Positive values are to be measured off above this and negative values below The line representing zero for Lead 2 is the vertical line in the center of the chart labeled e 2 Positive values are to be measured to the right of this negative values to the left

Suppose the values in the leads were e 1 = 16 e 2 = 10 and e 3 = −6 We should then take the horizontal line +16 and follow it across until it intersects with the vertical line +10 This point is crossed by a radius indicating +10 as the angle This is

to be compared with 8° which was obtained in the above example illustrating the use of Table XIII on page 411 When a value of less than 5 is to be dealt with as for example e 1 = 3 2 e 2 = 12 5 and e 3 = 9 3 it is better to multiply the values for e 1 and e 2 by 2 This will result in our measurements taking place in the outer zone where the radii do not lie so close together Accordingly for the above example we find that the horizontal line 6 4 (3 2 × 2) intersects the vertical line 25 (12 5 × 2) very near to the radius +75° This measurement is to be compared with the angle 76° derived from Table XII in the illustration on page 409 Again if e 1 = 8 e 2 = −4 and e 3 = −12 we will multiply 8 and −4 by 2 and find that the horizontal line +16 for e 1 intersects the vertical line −8 for e 2 very near to the radius −50° This is to be compared with the value −39 which was obtained by the use of Table XIII in the example on page 412

Dieuaide also offers a comparatively simple *table for determining the manifest value* (Em) From the fact that

$$Em = e \frac{1}{\cos \alpha}$$

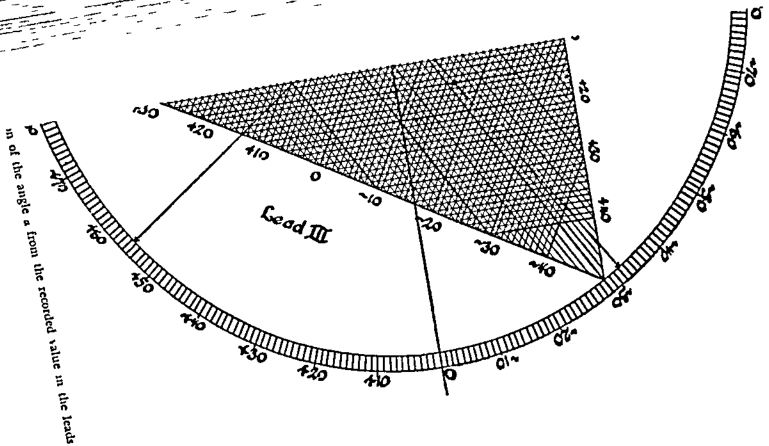
he has derived a table which follows

TABLE XIV

THE VALUE OF $\frac{1}{\cos \alpha}$

Angle α		$\frac{1}{\cos \alpha}$	Interpolation for 1	Angle α		$\frac{1}{\cos \alpha}$	Interpolation for 1
±0	±180	1 000	0 001	±30	±130	1 56	0 035
5	175	1 004	0 002	55	125	1 74	0 06
10	1 0	1 015	0 004	60	120	2 00	0 07
15	165	1 035	0 006	65	115	2 37	0 11
20	160	1 064	0 008	70	110	2 [illegible]	0 2
25	1 5	1 104	0 01	75	105	3 9	0 4
30	150	1 155	0 013	80	100	5 8	0 9
35	145	1 221	0 017	85	95	11 5	
40	140	1 305	0 027	90		1 155	0 013
45	135	1 41	0 03				

For α 90 use Lead s



ın of the angle a from the recorded value ın the leads

Fig 102 Dieuaide's chart for determining the direction of the electrical axis of any portion of the electrocardiogram by the measurement of the recorded value in Lead 1 (e 1) and Lead 2 (e 2)

to be compared with 8° which was obtained in the above example illustrating the use of Table XIII on page 411. When a value of less than 5 is to be dealt with as for example e 1 = 3.2, e 2 = 12.5 and e 3 = 9.3, it is better to multiply the values for e 1 and e 2 by 2. This will result in our measurements taking place in the outer zone where the radii do not lie so close together. Accordingly for the above example we find that the horizontal line 6.4 (3.2 × 2) intersects the vertical line 25 (12.5 × 2) very near to the radius +75°. This measurement is to be compared with the angle 76° derived from Table XII in the illustration on page 409. Again if e 1 = 8, e 2 = −4 and e 3 = −12, we will multiply 8 and −4 by 2 and find that the horizontal line +16 for e 1 intersects the vertical line −8 for e 2 very near to the radius −50. This is to be compared with the value −19° which was obtained by the use of Table XIII in the example on page 412.

Dieuaide also offers a comparatively simple *table for determining the manifest* value (Em). From the fact that

$$Em = e\frac{1}{\cos\alpha}$$

he has derived a table which follows

TABLE XIV

THE VALUE OF $\frac{1}{\cos\alpha}$

Angle α		$\frac{1}{\cos\alpha}$	Interpolation for 1	Angle α		$\frac{1}{\cos\alpha}$	Interpolation for 1
±0	±180	1.000	0.001	±50	±130	1.56	0.035
5	175	1.004	0.002	55	125	1.74	0.06
10	170	1.015	0.004	60	120	2.00	0.07
15	165	1.035	0.006	65	115	2.37	0.11
20	160	1.064	0.008	70	110	2.9	0.2
25	155	1.104	0.01	75	105	3.9	0.4
30	150	1.155	0.013	80	100	5.8	0.9
35	145	1.221	0.017	85	95	11.5	
40	140	1.305	0.022	90		1.155	0.013
45	135	1.41	0.03				

For 90 use Lead 3

To determine the manifest value from this table the value of Lead 1 should be multiplied by the figure which represents $\frac{1}{\cos \alpha}$ when the electrical axis has the direction indicated by the angle alpha shown in the two left hand columns The values are given at intervals of 5 degrees with interpolated values for one degree which can be used if needed Suppose for example Lead 1 = 5 and $\alpha = +65$ degrees Then $\frac{1}{\cos \alpha} = 2.37$ which multiplied by 5 gives 11 85 the value of E (For the angle $\alpha = +90$ degrees we cannot use Lead 1 as the factor because $\frac{1}{\cos \alpha}$ is infinity hence in such cases the value in Lead 3 is used)

In using either of these charts it is best to avoid dealing with small values it is better to multiply the whole series of figures by two in order to obtain measuring points farther apart This is particularly true in using Dieuaide s chart which confines the measurements to only two leads one of which may have a quite small deflection The chart of Carter Richter and Greene is perhaps the most satisfactory

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APPENDIX II

METHOD OF FILING AND INDEXING ELECTROCARDIOGRAPHIC RECORDS

To determine the manifest value from this table, the value of Lead I should be multiplied by the figure which represents $\frac{1}{\cos \alpha}$ when the electrical axis has the direction indicated by the angle alpha shown in the two left hand columns. The values are given at intervals of 5 degrees with interpolated values for one degree which can be used if needed. Suppose for example Lead I = 5 and $\alpha = +65$ degrees. Then $\frac{1}{\cos \alpha} = 2.37$ which multiplied by 5 gives 11.85, the value of E. (For the angle $\alpha = +90$ degrees we cannot use Lead I as the factor because $\frac{1}{\cos \alpha}$ is infinity, hence in such cases the value in Lead 3 is used.)

In using either of these charts it is best to avoid dealing with small values; it is better to multiply the whole series of figures by two in order to obtain measuring points farther apart. This is particularly true in using Dieuaide's chart which confines the measurements to only two leads, one of which may have a quite small deflection. The chart of Carter, Richter and Greene is perhaps the most satisfactory.

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APPENDIX II

METHOD OF FILING AND INDEXING ELECTROCARDIOGRAPHIC RECORDS

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METHOD OF FILING AND INDEXING ELECTROCARDIOGRAPHIC RECORDS

OWING to their size and character the preservation and filing of electrocardiographic records presents various difficulties. It seems worth while for this reason to give in detail the method which was gradually evolved at the New York Hospital before 1932 and which has given considerable satisfaction. It enables one to find promptly the record of any patient or to pick out with minimum effort a series of electrocardiographic records showing any peculiarity which has been diagnosed and indexed. The three strips Lead I, Lead 2 and Lead 3 are placed in order face downward and fastened together on the back with adhesive linen tape. Precordial leads may be placed in order below Lead 3 starting with the lead obtained farthest to the right. The precordial leads are folded behind the limb leads and the set is then placed in ordinary cardboard folders so that Lead 1 lies at the top. The name of the patient is written on the upper edge of the folder, the number of the record and the date on the lower flap. Subsequent records of the same patient may be placed in serial order in this folder and the number of the record and date noted on the outside so that all records of the same patient are readily found together. Folders are filed alphabetically according to names of patients. It has been found that after about ten years the cardboard of the folders becomes brittle and the folder must be replaced.

The legal size cardboard folder 9¾ by 15 inches was used because the great length of this container enabled us to file long strips of the record without folding. It has been found that when the record has been folded over it eventually cracks and tears at the point of folding. Another reason for using the long folder is that it gives space for a large number of filing signals which

ne letter to be placed along its edge It is possible, though to *use the ordinary letter size folder and by using signals of the* smallest size to find space for almost fifty diagnostic titles

A book is kept in which opposite the serial number of the record is written the name of the patient the age the clinical diagnosis the blood pressure and the amount of digitalis which had been given during the two weeks previous to taking the record Quinidine medication during the twenty four hours previous to the record is also noted These are the facts which the physician who interprets the record must have in mind in order to make his report as helpful as possible to the clinician Without them the report will not reveal its full usefulness

The accompanying table of diagnostic terms may be used in diagnosing the records or others may be substituted as desired

DIAGNOSIS TABLE

1 Normal sinus rhythm
2 a Sinus arrhythmia (R)
  b Sinoauricular block (O)
  c Simple bradycardia (P)
  d Ventricular escape (W)
3 Premature beats
  a Auricular (O)
  b Nodal (B)
  c Ventricular (R)
  d *Unusual premature beats (G)*
4 Simple tachycardia (P)
5 Paroxysmal tachycardia
  a Auricular (W)
  b Nodal (O)
  c Ventricular (B)
6 Auricular flutter
  a Paroxysmal (R)
  b Chronic (G)
7 Auricular fibrillation
  a Paroxysmal (P)
  b Chronic (W)
8 Auriculoventricular heart block
  a1 Partial block prolonged conduction } (O)
    2 Partial block dropped beats }
  b Complete block (B)
  c This prolonged conduction may be due to digitalis or disease of the bundle
9 Normal A V conduction time
10 Rhythm undiagnosed (R)
11 P Wave
  a Abnormally wide (G)
  b Notched (P)
  c Voltage unusually large (W)
  d Voltage unusually small (O)

e Unusual pecul arity (B)
ee In ersion of
12 QRS Group
a Abnormally wide (R)
bn Notched
bs Slurred } (O)
bn & s Notched and slurred
c Voltage unusually large (P)
d Voltage unusually small (W)
Unusual peculiarity of (O)
13 Right axis deviation of QRS (B)
14 Left axis deviation of QRS (R)
15 T wa e
a Prolonged (G)
b Notched (P)
c Voltage unusually large (W)
d Volt ge unusually small (O)
e Unus ial peculiarity } (B)
ee Unusual voltage of S T junction
f T wa e shows effect of digital s (R)
g Diphas c in Lead 1 (G)
h Dipha c in Lead 2 (P)
Diphasic in Leads 1 and 2 (W)
16 T wa e turned downward
a Lead 1 (O)
b Lead 2 (B)
c Leads 1 and 2 (R)
d Coronary T wave (C)
17 Bundle branch block (P)
18 Intraventricular block (W)
19 Neither right nor left axis de iat on
20 No abnormal ty of entricular wa es to suggest myocardial disease
21 This T wa e bnormal ty may be due to dig talis or to disease of the entricu lar muscle
a P obably the fo mer
b P obably the latter
22 These abnormal ties indicate diseas of the entr cular muscle
23 Ind ating dise se of ent icular muscle
24 Ind cating poo funct onal condit on of the muscle
25 The wa e are deformed by the presence of high skin resistance
26 The inte pr tation of this record is unsat sfactory d e to digitalis patient has rece d
27 Of doubtful significance
28 Of no lin al gnificance

It will be found that by combining the numbers of these diag noses with a very few words it will be possible to make a satis factory report of any electrocardiogram These diagnosis num bers are written by the physician in the book under the patient s name and serial number About 4 inches of each lead is torn off and the serial number and the number of the lead marked on the back In order that the secretary may know which way to

mount the pieces of the record it has been found satisfactory to turn the strip over as one would turn a check for endorsement and write the number and lead on the back of the left hand end of the strip The book is now turned over to the secretary who mounts the pieces of each lead and types the report of the record somewhat as follows A report in the book reads thus

4 rate 110 – 8 al (1 – R = 22) – 14 marked – 12 a bn 1 bs 2 and 3 d – 16 a – 22

It would be written as follows

Simple tachycardia rate 110 per minute
Partial auriculoventricular heart block with prolonged conduction time (P – R = 22 second)
Marked left axis deviation of QRS
QRS is abnormally wide and shows notching in Lead 1 and slurring in Leads 2 and 3
The QRS voltage is unusually small
The T wave is turned downward in Lead 1
These abnormalities indicate disease of the ventricular muscle

Again

7 (rate 95) – occas 3c – 13(12 bs 3 = 28) – 16 b – 21 – later record requested

This would be written as follows

Auricular fibrillation heart rate 95 per minute
Occasional ventricular premature beats
Right axis deviation of QRS
QRS group is slurred in Lead 3 but this is of no clinical significance
T wave turned downward in Lead 2 this T wave abnormality may be due to digitalis or to disease of the ventricular muscle
A later record is requested

This system saves a great deal of time in reporting the records but its chief value lies in the ease with which it adapts itself to indexing any peculiarities which may be considered important For this purpose colored signals are placed upon the upper edge of the folder the position and color of the signal indicating the peculiarity The Crallco vise signals which are of metal and clamp upon the edge of the folder have been found satisfactory cardboard strips may be used if pasted on Signals colored red green pink white orange and blue are used and these colors are repeated in this order from left to right so that 18 of the signals may be set edge to edge across the top of the folder In actual practice folders usually have but two or three signals and rarely as many as six A color key is made upon a piece of card

the colors being painted in their proper order for 15 inches and the number of the peculiarity to be recorded being marked below its corresponding color This card is used as a guide for placing the signals properly and also for picking out records with any desired peculiarity The features indexed in this way at the hospital are indicated in the diagnosis table by having the initial of one of the color signals in parentheses after it These are the features which seem especially important for such a cross index but obviously any other list of features could be used in this way

INDEX

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